DISSERTATION ON

A STUDY ON EPISTAXIS

Submitted in partial fulfillment of the requirements for

M.S. DEGREE BRANCH-IV OTORHINOLARYNGOLOGY

of

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003.

MARCH – 2010

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON EPISTAXIS"** submitted by **Dr. R. NANMULLAI,** appearing for **M.S. ENT.** Branch IV Degree examination in March 2010 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the **Tamil Nadu Dr. M.G.R. Medical University, Chennai.** I forward this to the **Tamil Nadu Dr. M.G.R. Medical University, Chennai**, **India**.

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ACKNOWLEDGEMENT

I would like to express my sincere gratitude to **Prof J MOHANASUNDARAM M.D, DNB,Ph D , The DEAN**, Madras Medical College, and Government General Hospital chennai, for having permitted me to use the hospital material in this study.

I am immensely grateful to **Prof. K. BALAKUMAR, M.S., D.L.O.,** The Director and Professor, Upgraded Institute of Otorhinolaryngology, for his valuable suggestions, encouragement and help in conducting this study.

I am immensely thankful to **Prof. A. MURALEEDHARAN, M.S., D.L.O.,** Professor, Upgraded Institute of Otorhinolaryngology, for his valuable guidance in conducting this study.

I am greatly indebted to **Prof. JACINTH.C.CORNELIUS, M.S., D.L.O.,** Professor, Upgraded Institute of Otorhinolaryngology, who encouraged and helped me throughout this study.

I am greatly thankful to **Prof. G.GANANATHAN, M.S., D.L.O.,** Professor, Upgraded Institute of Otorhinolaryngology for helping me in this study.

I express my sincere thanks to all the **Assistant Professors**, for their thoughtful guidance throughout the work.

I thank the **Secretary and Chairman of Institutional Ethical Committee**, Government General Hospital and Madras Medical College, Chennai.

I thank all the paramedical staff and other staff of the Upgraded Institute of Otorhinolaryngology for all their help and cooperation in conducting this study.

I thank all my colleagues and friends for their constant encouragement and valuable criticism.

Last but not least, I express my gratitude for the generosity shown by all the patients who participated in this study.

I am extremely thankful to my family members for their continuous support.

CONTENTS

INTRODUCTION

Epistaxis by definition is bleeding through the nose and is one of the most common and most difficult emergencies to treat. About 60% of people experience the episode atleast once in life time, with less then 10% of these requiring medical attention[18,19].

Most episodes are minor in nature and affected persons don't seek medical attention. In rare cases there may be massive epistaxis also. It gives much fear and anxiety of that of tumour and other non treatable conditions to the general population.

Epistaxis can have an anterior or posterior source and can be from septum or lateral nasal wall. A brief and pertinent history and physical examination generally determine the cause of bleeding.

Both systemic and local factors play a role. Hospital admission should be considered for patients with significant comorbid conditions or complications of blood loss.

Most of the cases can be managed conservatively with anterior nasal packing which exerts direct pressure over bleeding points.

AIMS OF THE STUDY

1. To find out the most common aetiology
2. Prevalence of epistaxis in each sex / age groups.
3. Relation and correlation of nasal anatomical variations with respect to epistaxis.
4. For early identification of the cause and thereby early management.

REVIEW OF LITERATURE

HISTORY OF EPISTAXIS

Epi-from above.

Staxis – drop by drop of fluid.

Bleeding through the nose.

Bleeding from the nose has been known from time immemorial. It has been mentioned in medical literature as early as 5th century B.C.

- **HIPPOCRATES** 5th century was the person first to note that pressure on the alae nasi was an effective method of controlling nose bleeds. He only first used cold fomentation and nasal packing to arrest bleeding[1].
- **ALI IBN RABBAN – AL-TABIRI** (AD 850) – in "The paradise of Wisdom" has written that "The complaint of nose bleeding is due to swelling of a vein and its rupture or perhaps a reduction in the force which confines the blood within"[1].
- **MORGAGNI (**1769) recognized 'The extremely turgid blood vessels about that part where the alae nasi are formed with the bone, about a finger's breadth more or less from the bottom of the nostril'. He has stopped nose bleeds by introducing his finger and 'pressing that part, whereupon the blood ceased to flow, so that it was not even discharged

by posterior nostril into the fauces'[1].

- **MORGAGNI** drew his inspiration from his teacher “VALSALVA” and for this reason little's area is called “LOCUS VALSALVAE”[1].

VALSALVA – Thought that the nasal bleeding was arterial in origin, and he stopped the bleeding by 'syringing the nose with cold water and applied spirit of wine to constrict the mouths of swollen arteries[1].

- **MAHOMED** (1880 – 81) described relation of hypertension with epistaxis in old age[1]. He stated that 'the frequency with which severe epistaxis occurs in old people with high arterial pressure is striking and for them very fortunate for if their noses did not bleed their brains would[1].
- **LITTLE** (1879) in 'Hospital Gazette' – described the caudal end of septum as the site of bleeding[1].
- **KIESSEL BACH** (1880) described similar observations, as LITTLE[1].
- **PILZ OF BRESSLAU** (1868) was the first to tie common carotid artery for epistaxis[1].
- **SEIFFERT** (1928) first described ligation of internal maxillary artery via transantral route for epistaxis[1].
- **GOOD YEAR** (1937) – first to tie anterior ethmoidal artery for epistaxis.
- **MARCUS** (1940) – not only revealed that endoscopy is helpful in

diagnosing epistaxis but also treated epistaxis by SPHENOPALATINE ARTERY LIGATION using endoscope[6].

- **LANGER AND TERRY –** demonstrated ANTERIOR ETHMOIDAL ARTERY LIGATION with nasal endoscopy[2].

VASCULAR ANATOMY OF NOSE

LAST (1978) CLASSIFICATION OF BLOOD SUPPLY OF NOSE[3]

TWO SYSTEMS OF ARTERIES SUPPLIES THE NOSE.

- **Internal carotid artery.**
- **External carotid artery.**

Internal carotid artery supplies nose via,

- Anterior ethmoidal artery.
- Posterior ethmoidal artery.

External carotid artery supplies nose via,

- Sphenopalatine artery – *Artery of Epistaxis.*
- Greater palatine artery.
- Superior labial artery.

INTERNAL CAROTID SYSTEM

Anterior ethmoidal artery

- Arises from the ophthalmic branch of the internal carotid artery.
- 2.4 cm posterior to anterior lacrimal crest.
- Supplies anterosuperior quadrant of the nasal septum and the lateral nasal wall.
- Varies in direct proportion to posterior ethmoidal artery.
- Absent unilaterally in 14%, bilaterally in 2.5%.

Posterior ethmoidal artery

- Arises from the ophthalmic branch of the internal carotid artery.
- 3.6 cm posterior to anterior lacrimal crest.
- Supplies posterosuperior quadrant of the septum and lateral wall of the nose.
- Dominant vessel of nose in embryonic life.
- If anterior ethmoid is absent, may arise directly from circle of Willis.
- Smaller than the anterior ethmoidal and absent in 20 – 30% of individuals[11,22].

EXTERNAL CAROTID SYSTEM

Sphenopalatine artery

- **Artery of epistaxis –** coronary artery of nose.
- Main arterial supply of nose.
- Arises from the 3rd part of the internal maxillary branch of the external carotid artery.
- Supplies posterior part of the lateral nasal wall and posteroinferior part of the septum.
- Comes out as 3 or more branches from sphenopalatine foramen into nasal cavity.

Greater palatine artery

•Arises from the 3rd part of the internal maxillary branch of the external carotid artery.

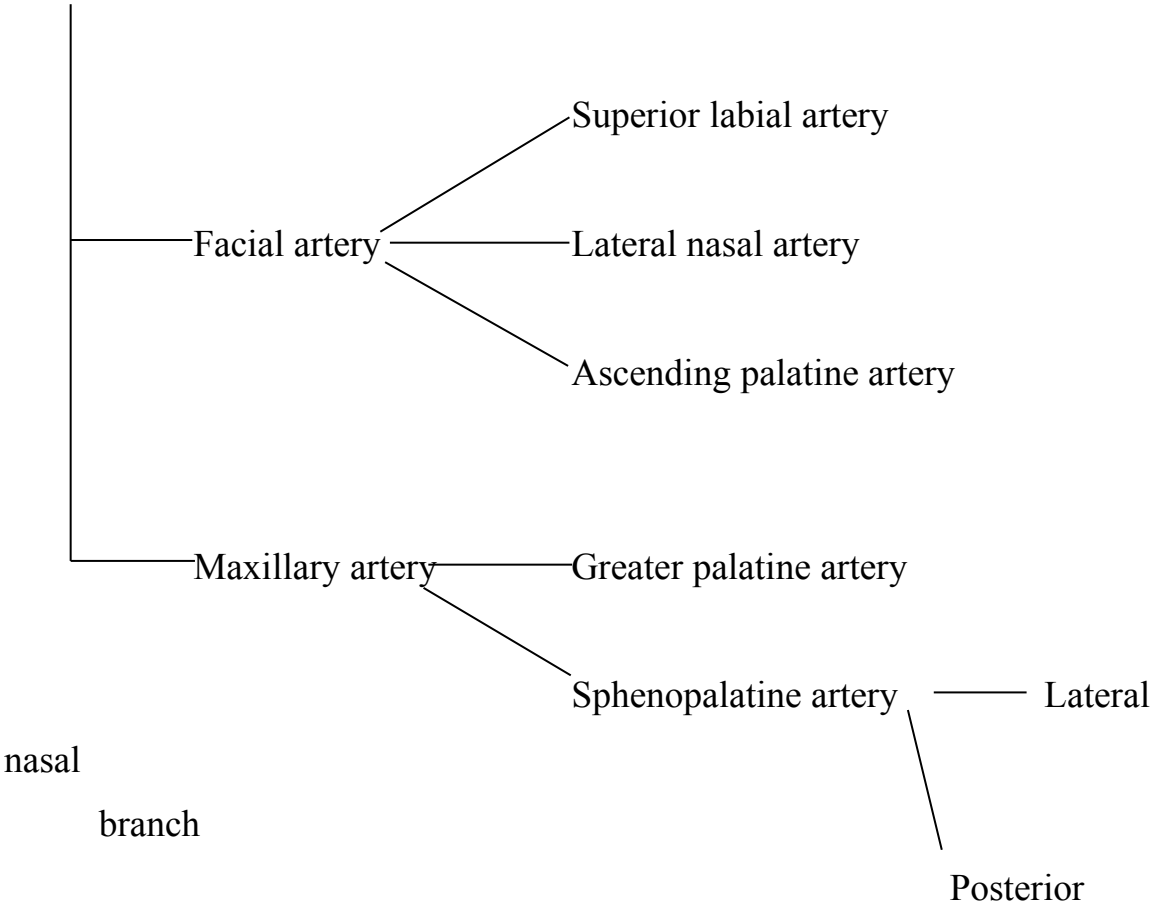
•Supplies anteroinferior quadrant of the nasal septum and lateral wall of the nose.

Superior labial artery

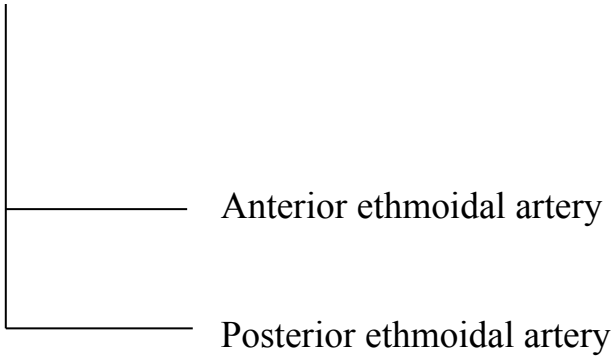
- Branch of the facial artery of the external carotid artery.
- Supplies anteroinferior quadrant of the nasal septum and lateral wall of the nose.

SUMMARY OF VASCULAR SUPPLY OF NASAL CAVITY

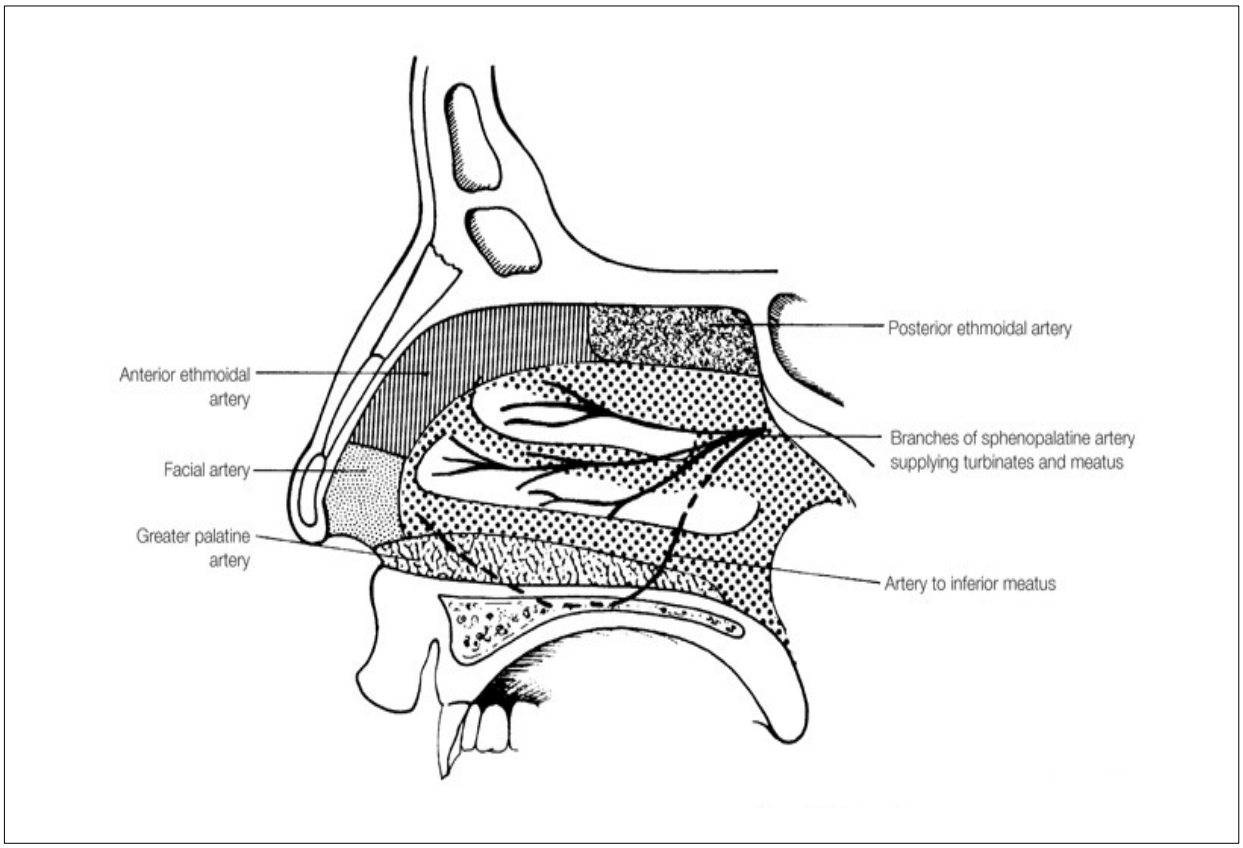
External carotid artery



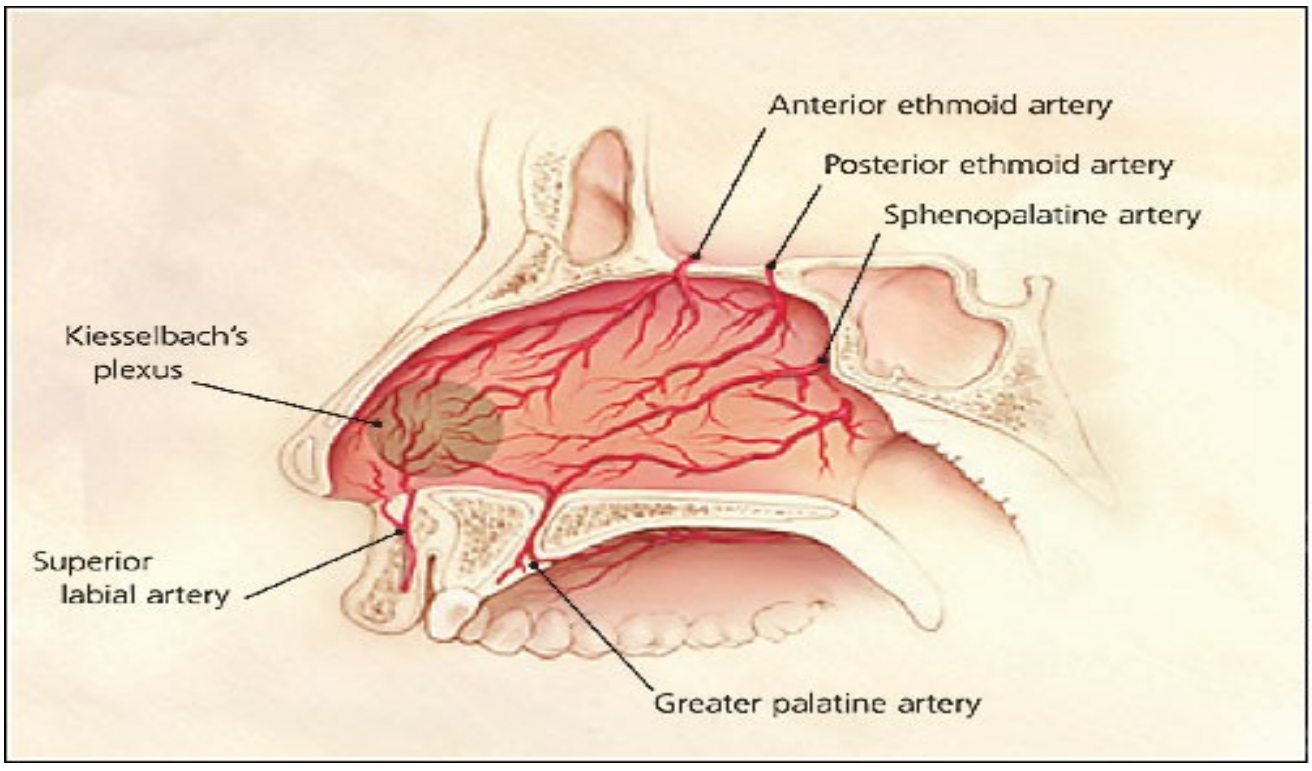
Internal carotid artery



ARTERIAL SUPPLY – LATERAL WALL OF NOSE



ARTERIAL SUPPLY – SEPTUM OF NOSE



MICROVASCULAR ANATOMY OF NOSE

Knowledge of the micro circulation of the nasal cavity is essential for the best management of epistaxis. But there are lot of anatomical variations in the microvascular anatomy of the nose particularly, the branching of the sphenopalatine artery – the artery of epistaxis[5].

Sphenopalatine artery is the most important arterial supply to nasal cavity (septum and lateral nasal wall). It enters through the **sphenopalatine foramen** which lies just inferior to the horizontal attachment of the middle turbinate and may be damaged in excessive enlargement of a middle meatal antrostomy. It divides into **posterior septal and posterior lateral rami.**

Posterior lateral division gives the **inferior and middle turbinate arteries** which runs in bony tunnels within the turbinates with reduced likelihood of that can be involved in epistaxis[9,10]. In the inferior meatus, the sphenopalatine branch dips below the level of the palate to re-emerge anteriorly, leaving the central portion of the meatus relatively avascular.

The **posterior septal branch** of the sphenopalatine artery runs medially across the face of sphenoid to the posterior part of the septum and supplies the posteroinferior septum and then takes an undulating course

anteroinferiorly in the mucoperichondrium.

The **greater palatine artery** enters the nasal cavity via the incisive canal and supplies the anteroinferior part of the septum and part of the lateral nasal wall adjacent to palate.

Nasal septal rami of the superior labial branch of the facial artery supplies most anterior part of the septum and contributes to kiesselbach's plexus. Lateral nasal artery, branch of the facial artery supplies vestibule and ascending palatine artery of the facial artery supplies a small area of the nasal cavity.

Anterior ethmoidal artery after its origin from the ophthalmic artery in the orbit passes between superior oblique and medial rectus muscle to the anterior ethmoidal canal and traverses the ethmoid and nasal cavities. It terminates in the region of the ethmoid fovea in a meningeal branch and a larger branch to the nasal roof, olfactory cleft and superior turbinate. It reaches the nasal cavity through the cribroethmoidal foramen and the cribriform plate and divides into the anterior nasal artery with superior, lateral and medial nasal branches, as well as a posterior branch. Anterior ethmoidal artery supplies anterosuperior part of the septum and the lateral nasal wall.

The **posterior ethmoidal artery** is smaller than the anterior

ethmoidal artery and is present in only 80% of individuals[11]. It passes above the superior oblique muscle to enter the posterior ethmoidal foramen situated 5mm anterior to the optic canal and 10 to 15mm behind the anterior ethmoidal foramen. It divides into a terminal meningeal branch and a branch to the posterosuperior nasal cavity (supplies posterosuperior portion of the nasal septum and the lateral nasal wall), olfactory sulcus and sphenoethmoidal recess.

There is **considerable overlap between the internal and external carotid arterial systems** on each side and between right and left side which may complicate attempts at arterial ligation in the management of epistaxis. Compensatory anastomotic flow via the facial arteries is thought to explain rebleeding which may occur following ligation or embolization.

There is a **sinusoid system in the nasal submucosa under autonomic control** on the most anterior septum and on the septum adjacent to the inferior turbinate and on the inferior and middle turbinates. This anterior septal tubercle was first described by **Morgagni**.

Clinically middle turbinate serves as a guide in deciding whether bleeding is from the internal or external carotid system and helps the surgeon to decide as to which artery has to be ligated.

SURGICAL ANATOMY OF THE SPHENOPALATINE FORAMEN

- Portal for the major arterial supply of the nasal cavity.
- Laterally lies the pterygopalatine space.
- Transmits the sphenopalatine artery, vein and the nasal palatine nerve.
- Key to the procedure of Endoscopic Sphenopalatine Artery Ligation (ESPAL).
- Crista ethmoidalis, a small bony projection lies anterior to the foramen in about 96% of cases, an important landmark during endoscopic procedure[29].

SPECIAL AREAS OF BLEEDING

Arterial bleeding

LITTLE'S AREA – James Little (1879)[8]

(or)

Kiesselbach's plexus (1880)[8]

(or)

Locus valsalvae

Caudal end of septum is the site where anastomosis of branches of the

Superior labial artery,

Nasopalatine artery,

Greater palatine artery,

Anterior ethmoidal artery and

Coronary artery of nose takes place.

Majority of bleeding occurs from here in children.

Bleeding from Little's area is more common because:

- Little's area is more vascular and it is covered by thin mucosa, so it is easily susceptible to injuries, particularly nose picking.
- Anatomical relationship to inspired air-Little's area is present in the anteroinferior part of septum above the level of the narrowest segment of nasal. passage – **Nasal valve.** At this point inspiratory

air flow is more rapid and directed towards septum. Rapid dry air causes inhibition of normal mucociliary action and in turn causes crust formation and bleeding.

Venous bleeding

1.Common in young persons.

2.Arises from vein which lies immediately behind the columella at the anterior edge of Little's area.

3.Mostly from **retrocolumellar vein.**

4.It runs vertically downwards and crosses the floor of the nose obliquely before joining the plexus on lateral wall of nose.

Haemorrhagic nodules

- Padgham and Parham (1993)[20].
- Consists of an aneurysmal dilatation of an unusually sited muscular artery with evidence of hypertensive changes in the wall and thrombus and haemorrhage in adjacent connective tissue.

Woodruff's plexus

- Woodruff (1949)[25].
- Naso-nasopharyngeal plexus.
- Collection of large blood vessels found in many people in the

lateral wall of the inferior meatus posteriorly.

- Appears to originate from the posterior pharyngeal wall.
- Venous in orgin.

Deviated Nasal Septum and Septal spur

Very few individuals have a perfectly straight septum. Most deviated nasal septums are asymptomatic.

The septal deformities may be classified into:

1. **Deviations**.

   Deviations are smooth deflections involving bony or cartilaginous portions or both. They are 'C' shaped or 'S' shaped.

2. **Spurs.**

   Sharp angulations in septum.

3. **Dislocations.**

Displacement of septal cartilage into nasal cavity.

The inspiratory air currents are often abnormally displaced and frequently become concentrated on small areas of nasal mucosa, producing an excessive drying effect. Crusting will then occur, and the separation of the crusts often produces ulceration and bleeding. The protective mucous layer may then be lost and resistance to infection reduced. The mucosa around a septal deviation may become oedematous as a result of **Bernoulli's** principle,

which states that when there is a flow of gas through a constriction lateral pressure drops which will, in turn, predispose to mucosal oedema in the affected area, thus further increasing the obstruction and leads to infection and *epistaxis.*

Septal turbinate

The septal turbinate represents an area (often visible on CT) of engorged vascular nasal mucosa on the septum. It may be unilateral or bilateral and can be a source of profound epistaxis. Its location may explain why a submucous resection cures some cases of septal epistaxis.

OTHER SITES OF BLEEDING

High in anterior part of septum.

Brown's area – posterior part of middle turbinate.

Inferior turbinates.

Lateral nasal wall.

Paranasal sinuses.

Nasopharynx.

Anterior cranial fossa when fractured.

VASCULAR PHYSIOLOGY OF NOSE

The dynamics of the nasal circulation depend to a large extent on the presence of **ARTERIOARTERIAL ANASTOMOSES** between the various arteries which contribute to the vascular supply of the nose. The branches of the anterior and posterior ethmoidal arteries join in a series of arcades in the upper one-third of the nose and the branches of the sphenopalatine artery anastomoses with those of the ethmoidal arteries above the level of the middle turbinate. Opposing heads of pressure meet in the anastomoses with a sharp interface between the two, which can be displaced by dropping the pressure in one or other of the opposing systems[1].

Shaheen (1967) demonstrated, by means of dye injection into the carotid vessels of live humans, that the dispersion of dye in the nasal mucous membrane could be affected by dropping the pressure in the system not being injected[10]. For instance, dye injected into the internal carotid artery failed to appear in the nose, confirming the poor circulation of the ethmoidal vessels, but when the external carotid was occluded at the time of injection the entire upper half of the nose was suffused with dye from above downwards. The rapidity with which such dye displacement takes place, confirms the importance of the arterioarterial anastomoses within the nose[1].

The importance of possible **ANASTOMOSES ACROSS THE MIDLINE** also must not be overlooked, either at the nasopharyngeal end or between the two anterior ethmoidal arteries at the crista galli. These observations could well explain the many documented reports of failed ligation in which surgeons assumed, probably incorrectly, that they had tied the wrong vessel simply because bleeding had not stopped after ligation[1].

The **ARTERIOVENOUS ANASTOMOSES** which are present at the anterior end of the inferior turbinate and septum at a microscopical level are probably of little importance in the aetiology and persistence of epistaxis, but their precise role remains unclear[1].

VASCULAR PATHOLOGY OF NOSE

Examination of the medium and smaller nasal arteries of persons dying in middle and old age has shown that these are subject to a progressive replacement of the muscle tissue in the tunica media by collagen (Shaheen, 1967)[10]. This change varies from interstitial fibrosis to almost complete replacement of the muscle by scar tissue. It seems that persons giving a history of epistaxis exhibit the more severe changes, but this is not to say that these changes are necessarily responsible for vessel rupture. They could, however, account for the lengthy duration of arterial haemorrhages, presumably because of a failure of the vessel to contract down in the absence of sufficient muscle in the tunica media[1].

It is also apparent that larger vessels of the calibre of the maxillary artery are prone to calcification (Monckeberg's sclerosis)[1]. The resulting lack of elasticity could be contributed to the pathogenesis of small vessel rupture by the creation of a local systolic hypertension.

The precise mechanism of bleeding is thought to be a dissecting aneurysm of the nasopalatine artery or one of its branches, but the factors initiating this process have, so far, not been identified.

It is also a mystery why bleeding should occur from the retrocolumellar vein in young subjects. Careful inspection of the site shortly after a bleed some times reveals a tiny area of local ballooning overlying the

vein, and this could possibly signify an area of vessel wall weakening, perhaps as a result of localized ischaemia and or trauma.

CLASSIFICATION OF EPISTAXIS

Structured clinical classification[30]

Primary	No proven causal factor.
Secondary	Proven causal factor.
Childhood	< 16 Years.
Adult	> 16 Years.
Anterior	Bleeding point anterior to piriform aperture.
Posterior	Bleeding point posterior to piriform aperture.

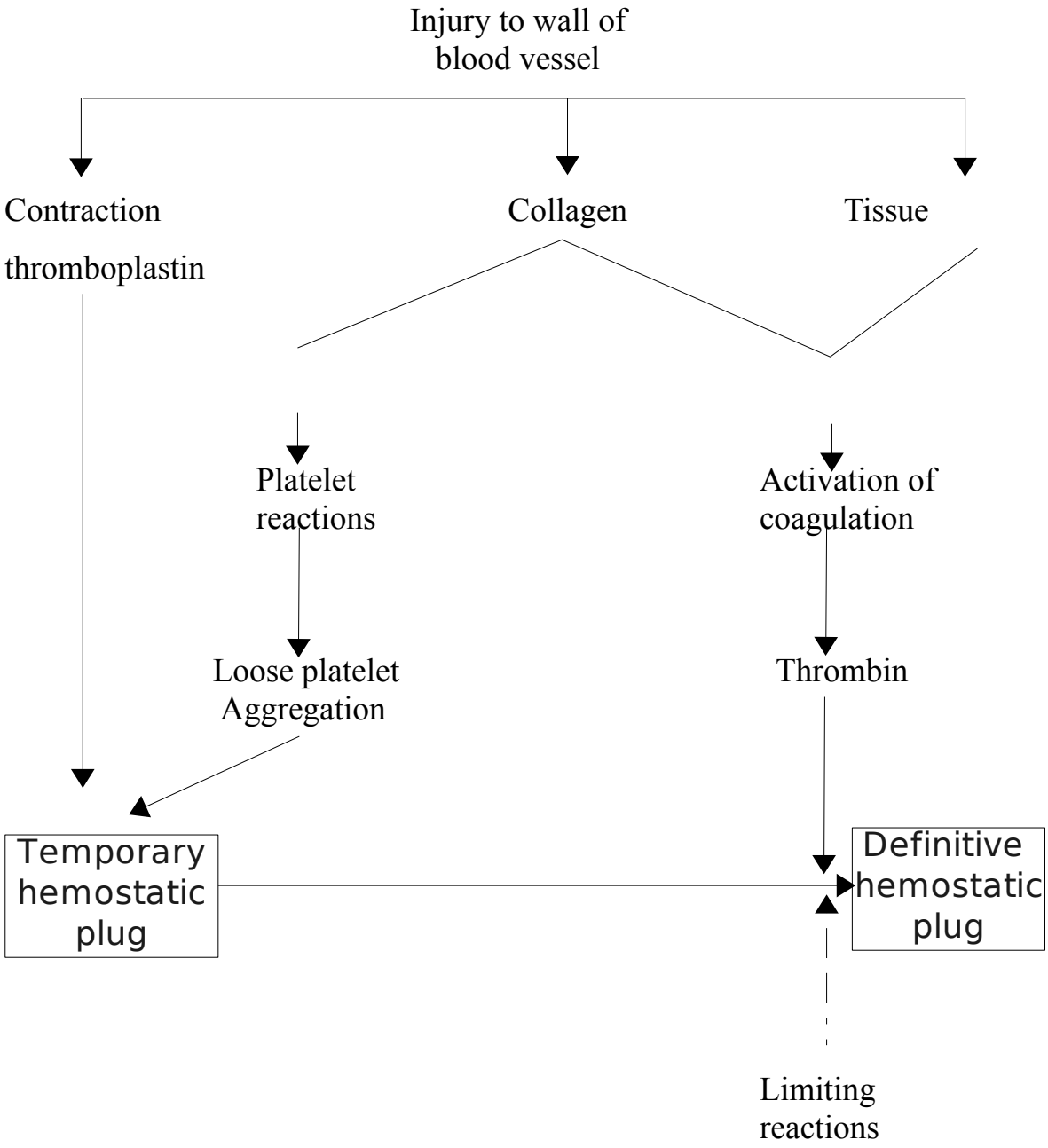
- **Anterior epistaxis:** Bleeding from a source anterior to the plane of the piriform aperture. This includes bleeding from the anterior septum and rare bleeds from the vestibular skin and mucocutaneous junction.
- **Posterior epistaxis:** Bleeding from a vessel situated posterior to the piriform aperture. This allows further subdivision into lateral wall, septal and nasal floor bleeding.
- Pearson(1983)[42] considered posterior epistaxis when bleeding point could not be located despite examination with a headlight, vasoconstrictors and suction, which is a clinically useful definition.
- Descriptive classification based on the severity and frequency of bleeding, Recurrent and acute, severe.

SYSTEM FOR NAMING BLOOD-CLOTTING FACTORS[41]

Factor[a]	Names
I	Fibrinogen
II	Prothrombin
III	Thromboplastin
IV	Calcium
V	Proaccelerin, labile factor, accelerator globulin
VII	Proconvertin, SPCA, stable factor
VIII	Antihemophilic factor (AHF), antihemophilic factor A, Antihemophilic globulin (AHG)
IX	Plasma thromboplastic component (PTC), christmas factor, Antihemophilic factor B
X	Stuart-power factor
XI	Plasma thromboplastin antecedent (PTA), antihemophilic factor C
XII	Hageman factor, glass factor
XIII	Fibrin-stabilizing factor, Laki-Lorand factor
HMW-K	High-molecular-weight kininogen, Fitzgerald factor
Pre-K_a	Prekallikrein, Fletcher factor
Ka	Kallikrein
PL	Platelet phospholipid

[a]Factor VI is not a separate entity and has been dropped.

RESPONSE TO INJURY41

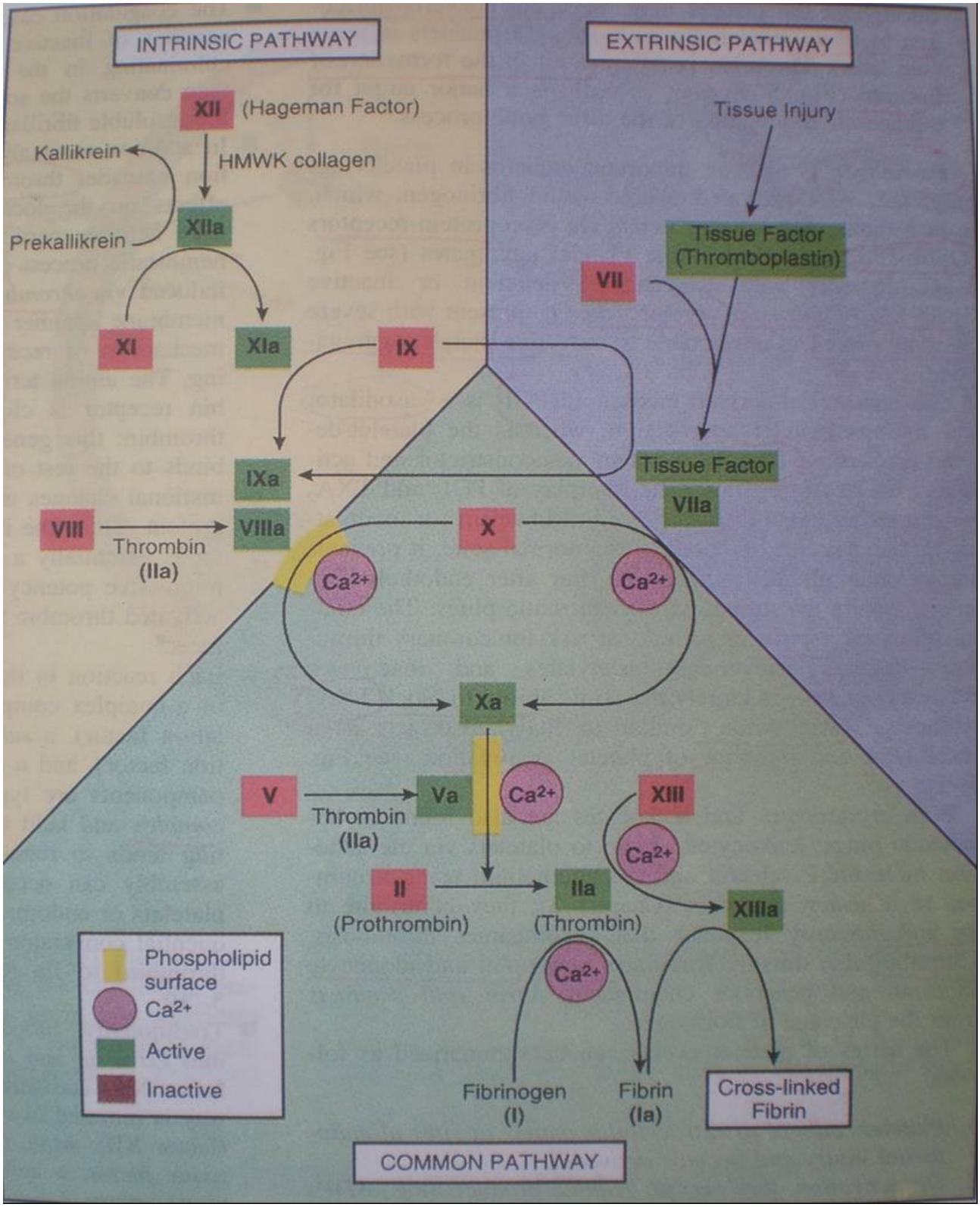


Summary of reactions involved in hemostasis.

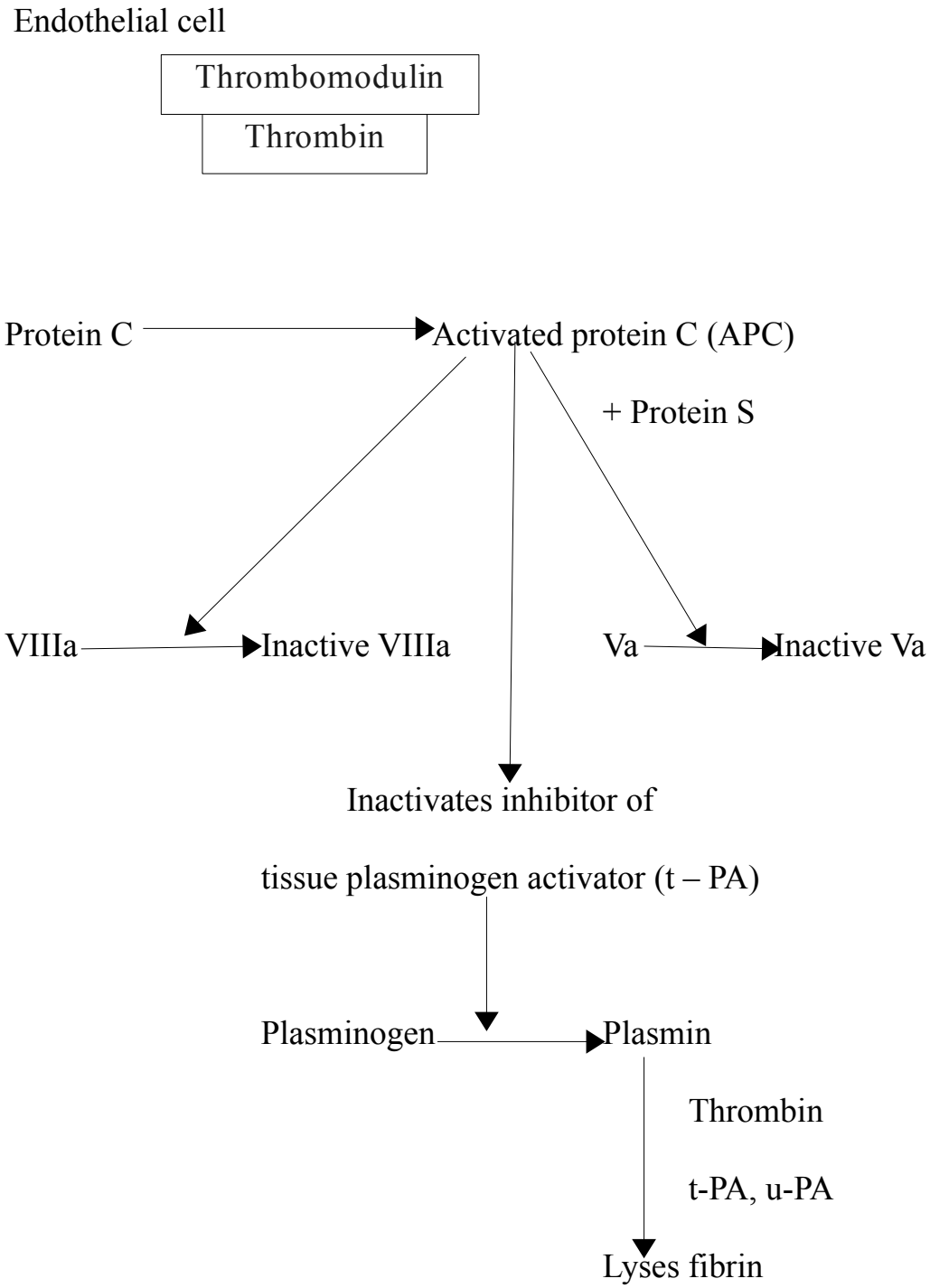
The dashed arrow indicates inhibition.
Injury when occurs initiates a series of events that lead to the formation of a clot (hemostasis) which seals off the damaged region that prevents further blood loss.

Initial event is constriction of the vessel and formation of a temporary hemostatic plug of platelets.

CLOTTING MECHANISM[40]



The clotting mechanism.



The fibrinolytic system and its regulation by protein C[41]

Anticlotting mechanism

AETIOLOGY OF EPISTAXIS

COMMONEST CAUSE BEING IDIOPATHIC.

Classified into[1,7]:

- Local Cause
- Systemic Cause

Local

Congenital

Unilateral choanal atresia

Meningocoele

Encephalocoele

Glioma

Acquired

Infective

Acute

Viral

Bacterial

Fungal

Chronic

Specific

Tuberculosis

Syphilis

Leprosy

Rhinoscleroma

Non specific

Ozaena

Inflammatory

Rhinosinusitis (allergic / vasomotor)

Nasal polyposis

Trauma

Iatrogenic

Facial trauma

Foreign body

Surgery

Idiopathic

Little's area,

Superior part of nose,

Middle meatus,

Woodruff's plexus

Neoplastic

Benign

Transitional cell papilloma,

Angiofibroma,

Others.

Malignant

Squamous cell carcinoma,

Adenocarcinoma,

Adenoid cystic carcinoma,

Olfactory neuroblastoma,

Lymphoma.

Drug-induced

Rhinitis medicamentosa (topical decongestants / cocaine)

Inhalants

Tobacco,

Cannabis,

Heroin,

Chrome,

Mercury,

Phosphorus,

Wood dust.

Systemic

1. **Bleeding disorders**

- **Coagulopathies**
  - **Inherited**

    Coagulation factor deficiencies,

    i.e. factor VIII (haemophilia A,B) and factor IX deficiency.
  - **Acquired**

    Anticoagulants,

    Liver disease,

    Vitamin K deficiency

    Disseminated intravascular coagulation (DIC)

    Acquired inhibitor.
- **Platelet disorders**
  - **Thrombocytopenia**

    Congenital

    Acquired

    Marrow failure,

    Aplasia,

    Drugs,

    Infiltration,

    Increased consumption

    Immune,

DIC,

Hypersplenism,

Massive blood loss.

■ **Platelet dysfunction**

congenital

Von Willebrand's disease

Bernard Soulier syndrome

Glanzmann's thrombasthenia

Acquired

Myeloproliferative disease

Leukaemia

Uraemia

Dysparaproteinaemias

Drugs : Aspirin, NSAID's

Acquired storage pool disease

i.e. bypass.

- **Blood vessel disorders**

Congenital

Osteogenesis imperfecta

Hereditary haemorrhagic telangiectasia

Acquired

Amyloid

Vasculitis

Vitamin C deficiency

- **Hyperfibrinolysis**

Congenital

α_2 antiplasmin deficiency

Acquired

Malignancy

DIC

Fibrinolytic therapy i.e. streptokinase.

2.**Drugs**

Aspirin

Anticoagulants

Chloramphenicol

Methotrexate

Immunosuppressives

Alcohol

Dipyridamole

3. **Neoplasms**

4. **Idiopathic**

Inflammatory disorder

Sarcoidosis

Wegener's granuloma

Lethal midline granuloma

5. **Others**

Liver failure

Hypothyroidism

Cardiovascular conditions that increase venous pressure (Congestive heart failure, mitral valve stenosis)

Hypertension (unproven relationship)[14]

HIV

EPISTAXIS TRAY

SUPPLIES AND EQUIPMENT FOR THE EVALUATION AND TREATMENT OF EPISTAXIS IN THE URGENT CARE SETTING

1. Personal protective supplies

    Mask

    Gown

    Eye protection

- Wall suction and tubing
- Frazier tip suctions, size 10 and 12 French
- Headlight
- Nasal speculum
- Bayonet forceps
- Cotton or neurosurgical cottonoid sponges
- Silver nitrate cautery sticks
- Packing materials

    Merocel nasal sponges (Merocel Corporation)

    Vaseline strip gauze (Sherwood medical)

    Gelfoam (Upjohn company)

Surgicel (Johnson & Johnson)

- Suction cautery unit
- 0- and 30- degree rigid nasal endoscopes with light source
- Optional loupe magnification
- Vasoconstrictors and anesthetics

  4% or 10% cocaine (combination anesthetic / decongestant)

  0.5% to 1% phenylephrine

  2% topical lidocaine solution

  1% injection lidocaine with 1:100,000 epinephrine
- Antibiotic ointment (without neomycin)
- Tongue blades
- Two 10-cc syringes with 18- and 27- gauge needles

- Epistaxis tray helps to attend cases immediately, to take emergency measures and to treat the patients effectively.
- Avoids delay in attending patients.

INVESTIGATIONS OF EPISTAXIS PATIENT

Depends upon provisional diagnosis.

1. Complete Haemogram.
2. Blood Grouping and Typing.
3. Bleeding Profile.
4. Diagnostic Nasal Endoscopy.
5. Serum Biochemistry (Urea / Sugar / Electrolytes).
6. Urine Analysis.
7. Liver Function Test.
8. X-ray Paranasal Sinuses – Water's View.
9. CT Paranasal Sinuses and Brain – Plain and Contrast.
10. Electro Cardiography.
11. Chest X-ray PA View / CT scan.
12. Angiography / Digital Substraction Angiogram.
13. MRI – Paranasal Sinuses / Brain / Angiogram.

TREATMENT OF EPISTAXIS

Depends on cause, site and severity of bleeding

TREATMENT CONSISTS OF

General treatment.

Local treatment to stop bleeding.

Treatment of specific cause of bleeding.

Arterial ligation – rarely.

GENERAL TREATMENT

Reassurance

Bed rest

Sedation

Hospitalisation

Treatment of Shock – Resuscitation

1. Fluid replacement – crystalloids, colloids.
2. Blood Transfusion – if packed cell volume below 25%.

Antibiotic – for prevention of secondary infection.

Haemostatics

LOCAL TREATMENT TO STOP BLEEDING

1. **Temporary pressure** over the alae nasi by pinching the nose, while sitting upright.
2. **Anterior Nasal Packing :**
    1. Vaseline Gauze
    2. Gelfoam
    3. Merocel, etc.,
3. **Postnasal Packing** if necessary
    1. Gauze
    2. Foley's catheter
    3. Balloon packs, etc.,
4. **Cauterization of bleeding point**

    Chemical
    - 1% Silver nitrate
    - 40% Trichloracetic acid
    - Copper sulfate
    - Carbolic acid, etc.,

    Diathermy
5. **Direct Therapies**

    Bipolar diathermy

    Chemical cautery

    miniature targeted packs

Endoscopy control – bipolar diathermy

6. **Indirect Therapies**

Nasal packing	– ribbon gauze with petroleum jelly or bismuth iodoform paraffin paste (BIPP)
Merocel and kaltostat	– special tampons
Balloon catheters	
Hot water Irrigation[15]	– water at 50 degree C
Systemic medical therapy	– Tranexamic acid – Epsilon Amino Caproic Acid (EACA)

7. **Surgical Management**

- Posterior packing;
- Ligation techniques;
- Septal surgery techniques;
- Embolization techniques;

Complications of nasal packing – ulcerations and abrasions, discomfort to the patient, synechiae, sinusitis, toxic shock syndrome, alar necrosis, septal or palatal necrosis, hypoxemia, apnea and cardiac arrhythmias requiring humidified oxygen, cardiac and hemodynamic monitoring.

TREATMENT OF SPECIFIC CAUSE

The specific cause of bleeding must be identified and treated promptly.

a) **MEDICAL – MOSTLY FOR SYSTEMIC CAUSES**

b) **SURGICAL – MOSTLY FOR LOCAL CAUSES**

ARTERIAL LIGATION

For severe epistaxis where causes are not known.

DIRECT APPROACH

External carotid artery.

Internal maxillary artery.

Anterior ethmoidal artery can be ligated – As adjuvant procedure or in cases of confirmed ethmoidal bleeding (Ethmoidal #, Iatrogenic tear).

ENDOSCOPIC APPROACH

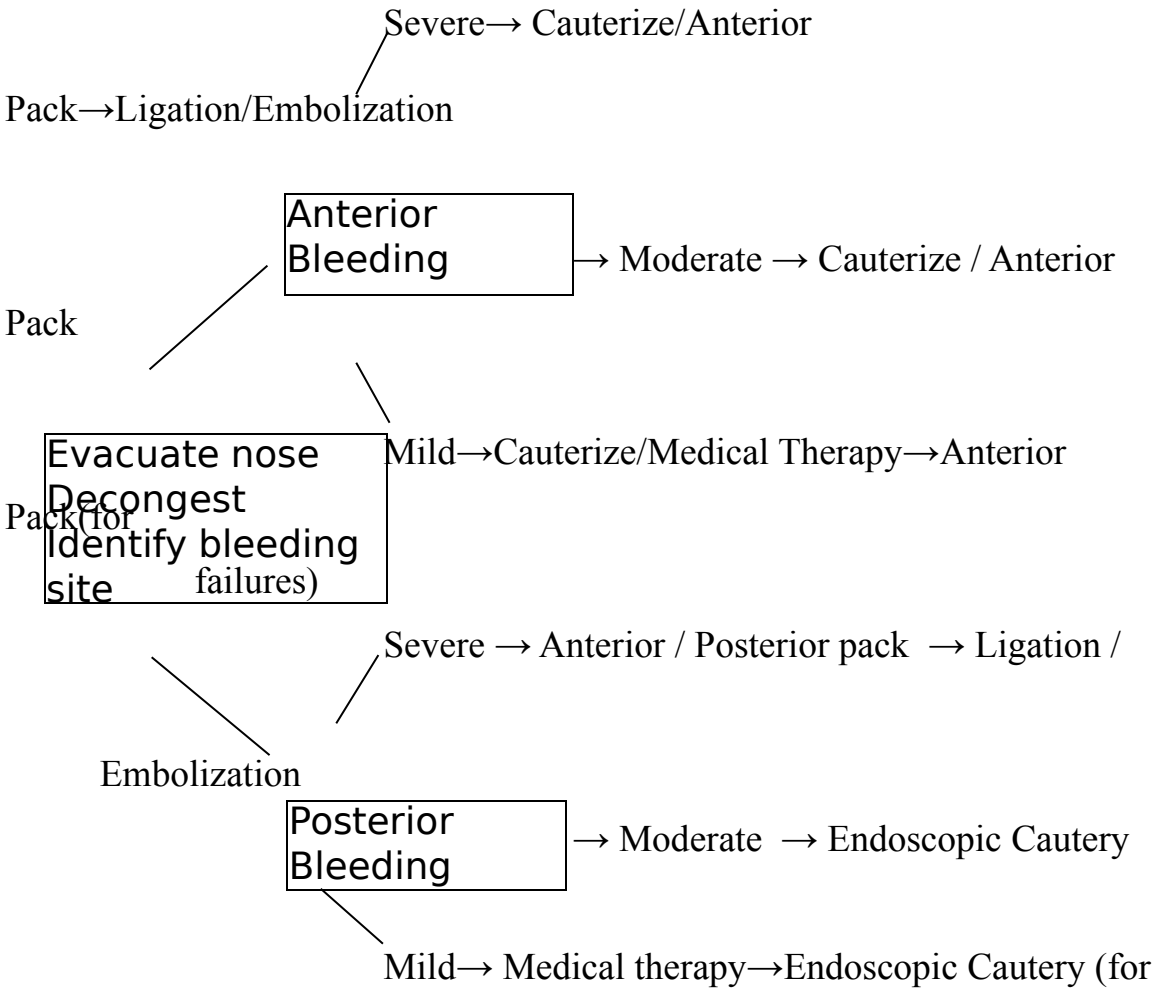
Sphenopalatine artery ligation[27,29].

Anterior ethmoidal artery can be ligated[38].

ENDOSCOPIC MANAGEMENT OF EPISTAXIS CONSISTS OF

1. Endoscopic cauterization.
2. Endoscopic septal surgery.
3. Endoscopic sinus surgery.
4. Endoscopic removal of mass, foreign body.
5. Endoscopic ligation of vessels.
6. Assistance in laser, cryo surgeries and other surgeries.

Management Protocols for acute epistaxis



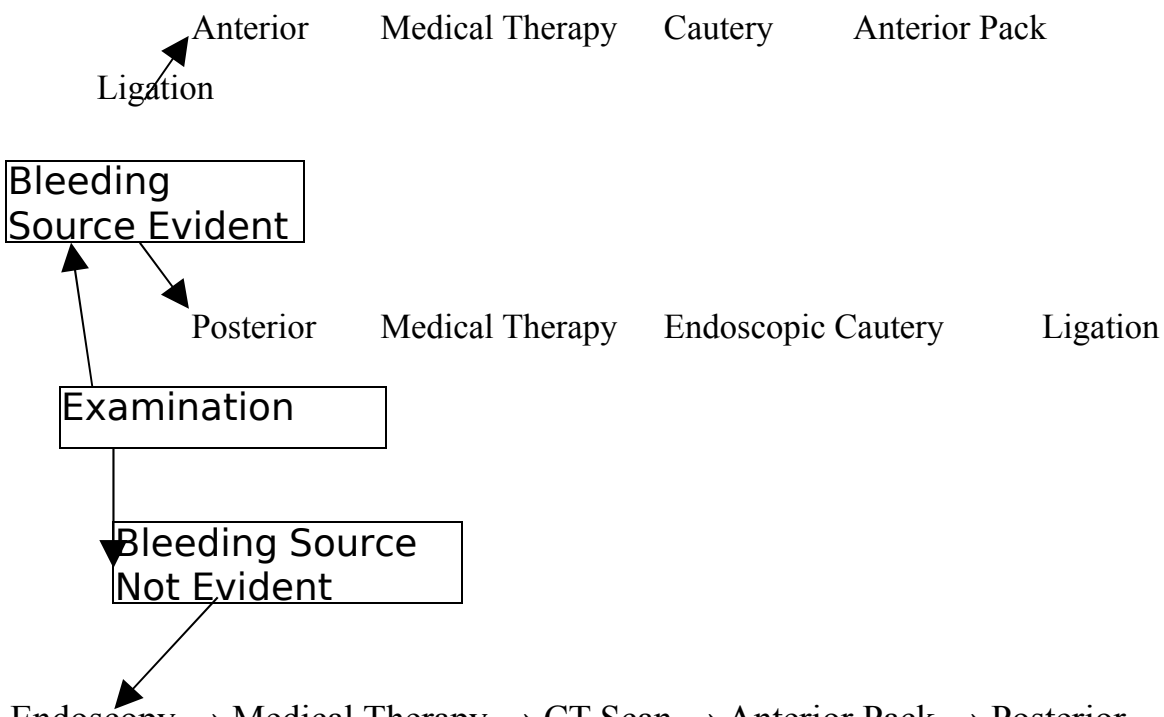
Conservative treatment

Medical - For mild, recurrent without active bleeding

- Minimize or eliminate the initiating or exacerbating factors, limiting trauma.
- Nasal hydration.
- Hematological assessment.
- Hippocratic technique – advice to pinch nostril.

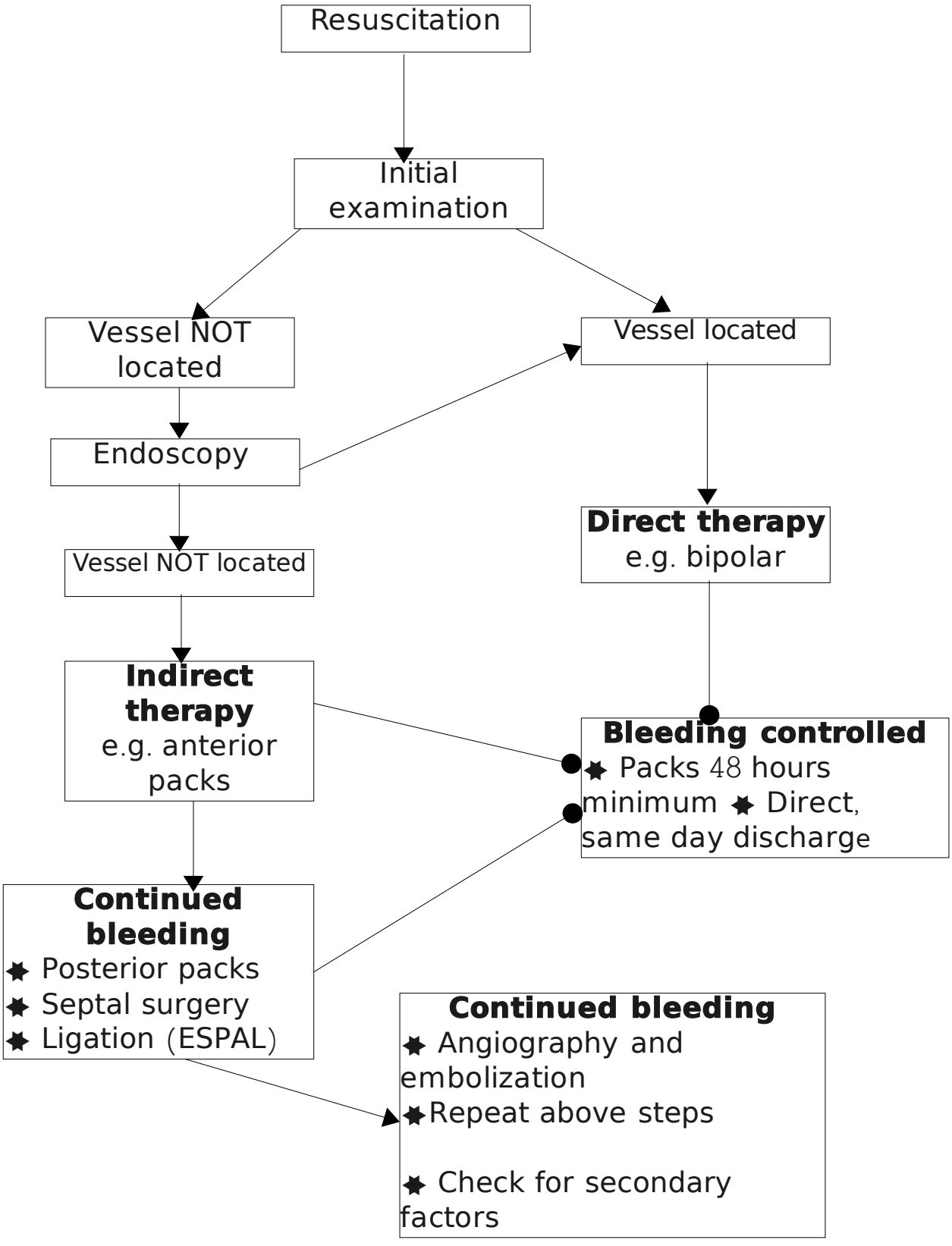
Anterior Nasal Cautery-to control acute anterior epistaxis -chemical or thermal.

Management protocols for chronic or recurrent epistaxis[23]

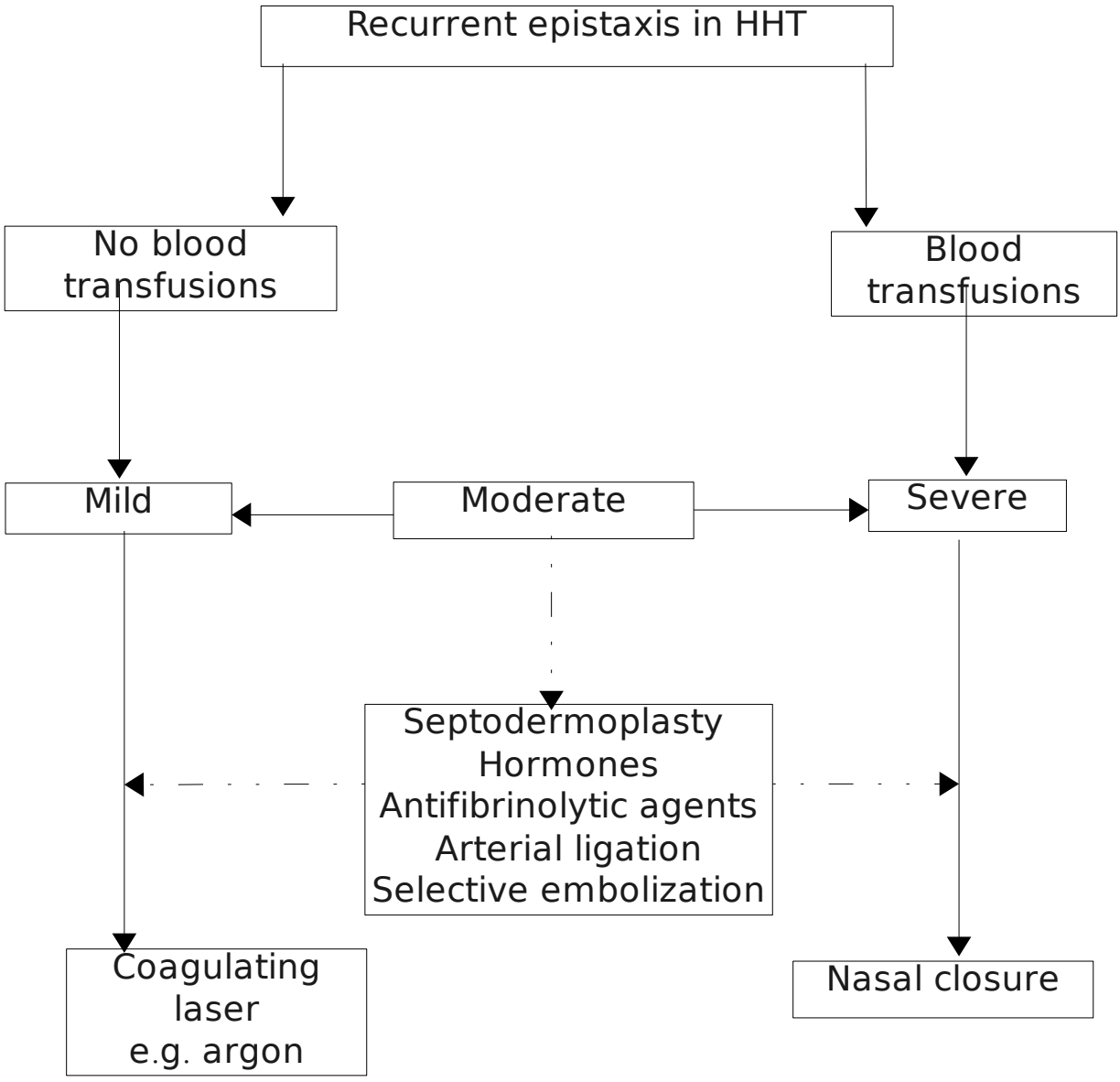


Endoscopy → Medical Therapy → CT Scan → Anterior Pack → Posterior Pack → Ligation

MANAGEMENT STRATEGY FOR ADULT PRIMARY EPISTAXIS[23]



TREATMENT ALGORITHM FOR HEREDITARY HAEMORRHAGIC TELANGIECTASIA[16].



MATERIAL AND METHODS

Study design – Prospective

Study period – July 2007 to September 2009

Study place – Upgraded institute of otorhinolaryngology, Government General Hospital, Madras Medical College, Chennai.

Patients above 12 years who reported with history of bleeding through the nose were examined and full head and neck examination done after elaborating detailed history.

- Examined to identify whether anterior or posterior bleeding and to identify the bleeding source.
- If bleeding was severe, anterior nasal packing and posterior nasal packing if necessary was done and all patients were admitted in the ward.
- Complete blood investigations and other relevant investigations like DNE, X-Ray, PNS/Chest, CTPNS, ECG etc done.
- For the patients for whom no active bleeding or source identified at the time of examination were advised relevant investigations, treatment and followup.
- 120 cases were included in this study from the ENT ward and out patient department from July 2007 – Sep 2009, UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY, Government

General Hospital and Madras Medical College, Chennai.

Inclusion Criteria

1. Age group above 12 years
2. History of bleeding through the nose

Exclusion Criteria

1. Age below 12 years
2. Patients who are not willing for study

No attempt was made to assess the nose of bleeding patients in severe epistaxis as immediate care was the primary requirement.

Traumatic epistaxis with poly trauma was excluded to give attention to immediate care of the patient to rule out and treat injury to other vital structures.

Patients with life threatening emergencies (ie myocardial infarction etc.)

Endoscopes : 0 and 30 degree, 4 mm rigid nasal endoscopes (Serwel and storz) camera and light source and necessary instruments.

Anaesthesia : 4% Xylocaine topical and decongestant

Packing : Vaseline pack, merocel pack, post nasal pack, foley's catheter.

OBSERVATIONS

Table 1

AETIOLOGY OF EPISTAXIS

S. No	Etiology	Male	%	Female	%	Total	Total %
1	Idiopathic	31	25.83	25	20.83	56	46.67
2	Trauma / RTA	6	5	4	3.33	10	8.33
3	DNS / Spurr	1	0.83	2	1.67	3	2.5
4	Chr.Sinusitis / DNS	2	1.67	3	2.5	5	4.17
5	JNA / Rec. JNA	9	7.5	0	0	9	7.5
6	Rhinosporidiosis	3	2.5	0	0	3	2.5
7	Polyps / Sinonasal Polyposis	0	0	3	2.5	3	2.5
9	Septal Perforation	1	0.83	0	0	1	0.83
10	Rhinolith	1	0.83	0	0	1	0.83
11	Malignant Growth	5	4.17	1	0.83	6	5
12	Bleeding Polyposis	1	0.83	2	1.67	3	2.5
13	Chronic sinusitis	0	0	2	1.67	2	1.67
14	DNS	1	0.83	2	1.67	3	2.5
15	Granulation Tissue Lt Septum	1	0.83	0	0	1	0.83
16	Hypertensive	4	3.33	2	1.67	6	5
17	Benign mass / Inv. Papilloma	0	0	2	1.67	2	1.67
18	Haemangioma	1	0.83	0	0	1	0.83
19	Iatrogenic	2	1.67	0	0	2	1.67
20	Coagulation Deficits	3	2.5	0	0	3	2.5

DNS – Deviated nasal septum.JNA – Juvenile nasopharyngeal angiofibroma

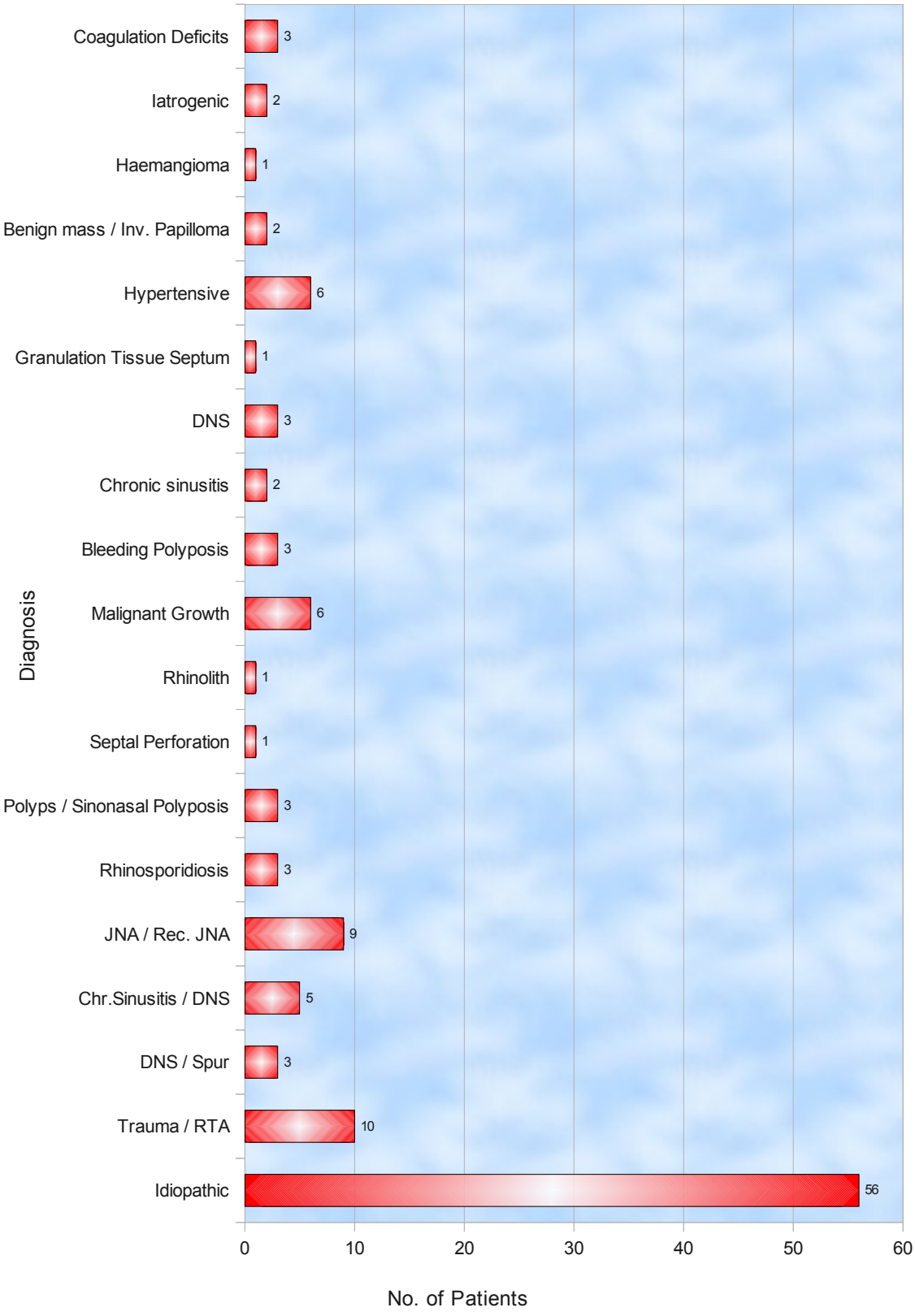
The most common cause in this study is idiopathic (46.67 %).

Other causes noted are trauma(8.33%), DNS with Spur and sinusitis(10.84%), polyps(5%), benign(1.76%) and malignant(5%) growth. Hypertension, rhinosporidiosis, hematological causes are noted.

Haemangioma, rhinolith, septal perforation and iatrogenic causes have also been noted.

JNA has been noted exclusively in adolescent males.

AETIOLOGY OF EPISTAXIS



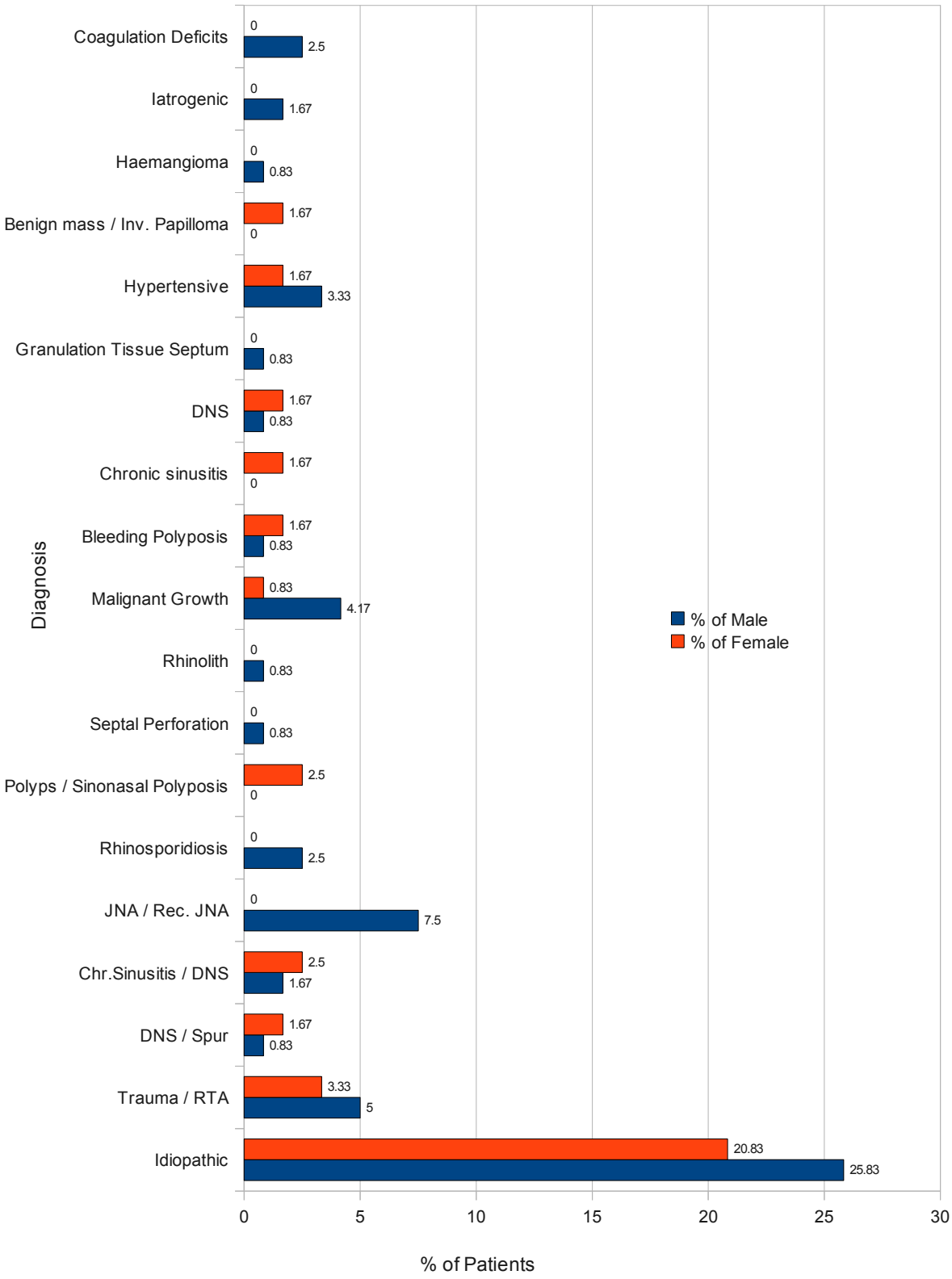
AETIOLOGY OF EPISTAXIS

MALE AND FEMALE (%)

Table 2

Aetiology	% of Male	% of Female
Idiopathic	25.83	20.83
Trauma / RTA	5	3.33
DNS / Spur	0.83	1.67
Chr.Sinusitis / DNS	1.67	2.5
JNA / Rec. JNA	7.5	0
Rhinosporidiosis	2.5	0
Polyps / Sinonasal Polyposis	0	2.5
Septal Perforation	0.83	0
Rhinolith	0.83	0
Malignant Growth maxilla	4.17	0.83
Bleeding Polyposis	0.83	1.67
Chronic sinusitis	0	1.67
DNS	0.83	1.67
Granulation Tissue Septum	0.83	0
Hypertensive	3.33	1.67
Benign mass / Inv. Papilloma	0	1.67
Haemangioma	0.83	0
Iatrogenic	1.67	0
Coagulation Deficits	2.5	0

Aetiology of Epistaxis



COMMONEST CAUSES IN EACH AGE GROUPS

Table 3

Age Group	Males	Females
13 – 20	JNA/Idiopathic	Idiopathic
21 – 30	Idiopathic	Idiopathic / Trauma / DNS, Sinusitis
31 – 40	Idiopathic / Trauma	Idiopathic / Sinusitis
41 – 50	Idiopathic / Hypertension	Polyps / Malignancy
51 – 60	Malignancy / Idiopathic	Hypertension / Idiopathic
61 – 70	Malignancy / Idiopathic	-

The most common cause of epistaxis in this study is idiopathic(46.67%), in both sexes. (Male 25.83%, Female 20.83%)

Other common causes in this study are trauma (8.33%), DNS with chronic rhinosinusitis and DNS with spur (10.84%) and polyps (5%).

JNA is exclusively noted in second decade males, malignancy (5%) and hypertension has been noted in older age group.

Trauma has been noted mainly in 13 to 30 age group.

DISTRIBUTION-AGE GROUP

Table 4

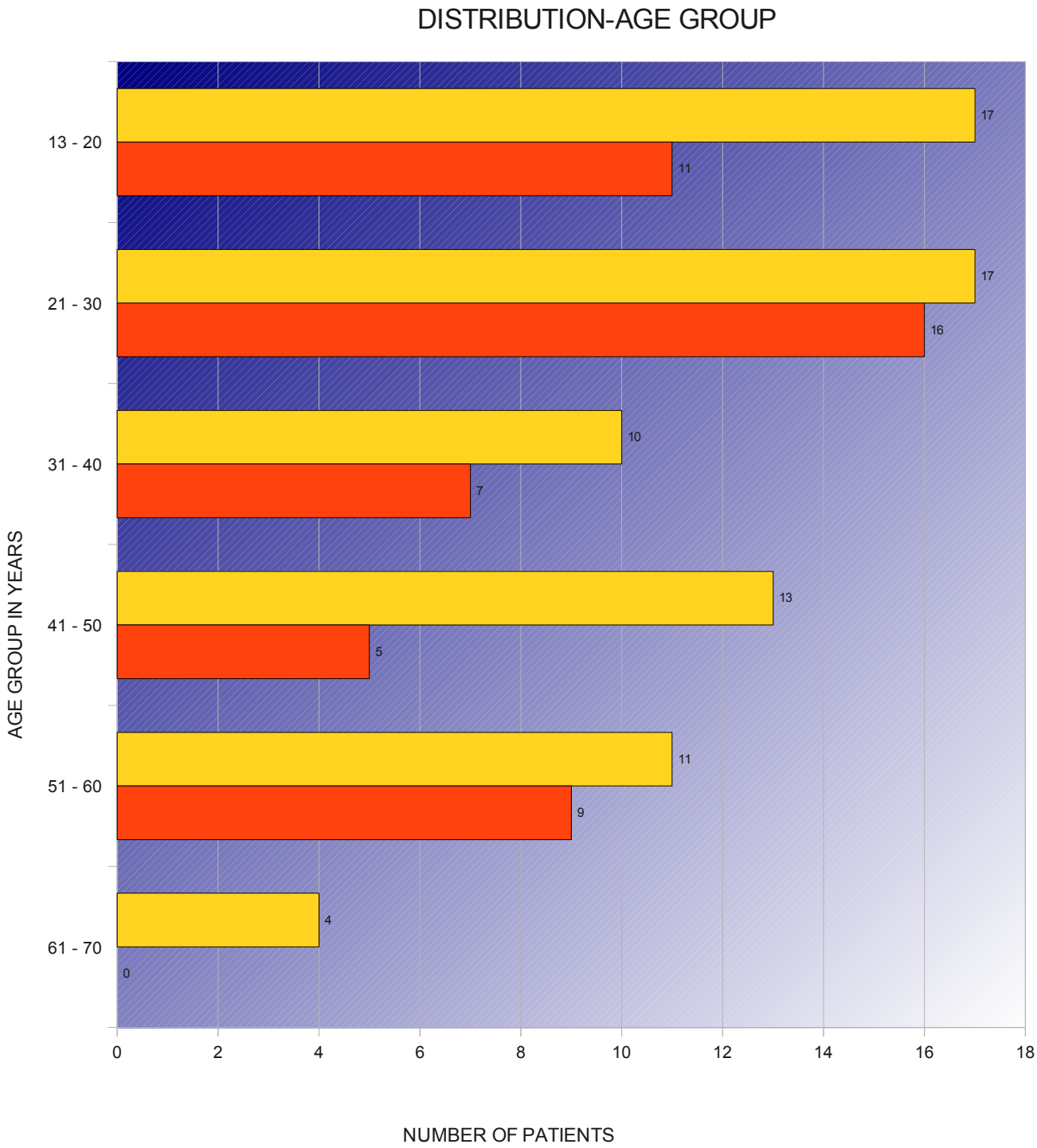
S. No	Age Group in Years	Male	Female	Total	%
1	13 - 20	17	11	28	23.33
2	21 - 30	17	16	33	27.5
3	31 - 40	10	7	17	14.17
4	41 - 50	13	5	18	15
5	51 - 60	11	9	20	16.67
6	61 - 70	4	0	4	3.33

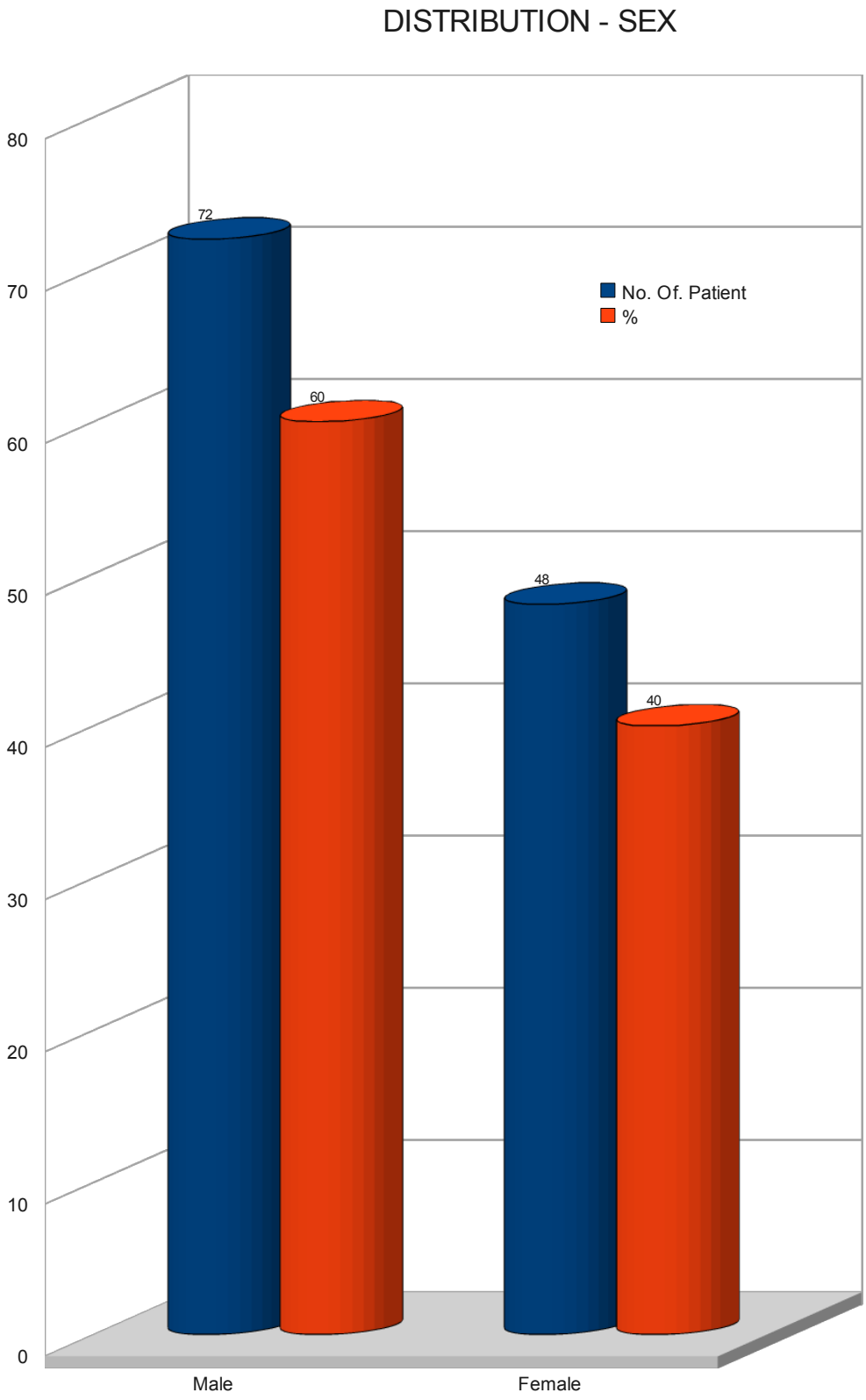
- The symptom of nose bleeding is more commonly noted in this study in 21 to 30 (27.5 %), 13 to 20 (23.33 %) age groups.
- The next most frequently involved group is 51 to 60 (16.67 %), 41 to 50 (15 %) age group.
- JNA is exclusively noted in second decade adolescent males.

DISTRIBUTION-SEX

Table 5

S.No	Sex	No. Of. Patient	%
1	Male	72	60
2	Female	48	40
	Total	120	100


DISTRIBUTION-AGE GROUP
AGE GROUP IN YEARS
13 - 20
21 - 30
31 - 40
41 - 50
51 - 60
61 - 70
17
11
17
16
10
7
13
5
11
9
4
0
0
2
4
6
8
10
12
14
16
18
NUMBER OF PATIENTS


DISTRIBUTION - SEX
80
70
60
50
40
30
20
10
0
72
60
48
40
No. Of. Patient
%
Male
Female

DISTRIBUTION-SEX - AGE GROUP

Table 6

Age Group in Years	% of Male	% of Female	Total %
13 - 20	14.17	9.17	23.33
21 - 30	14.17	13.33	27.5
31 - 40	8.33	5.83	14.17
41 - 50	10.83	4.17	15
51 - 60	9.17	7.5	16.67
61 - 70	3.33	0	3.33

- Males are more commonly affected in this study(60%).
- Males are commonly affected more in almost all age groups.
- JNA is exclusively noted in adolescent males in this study.

ANATOMICAL VARIATIONS ASSOCIATED WITH EPISTAXIS

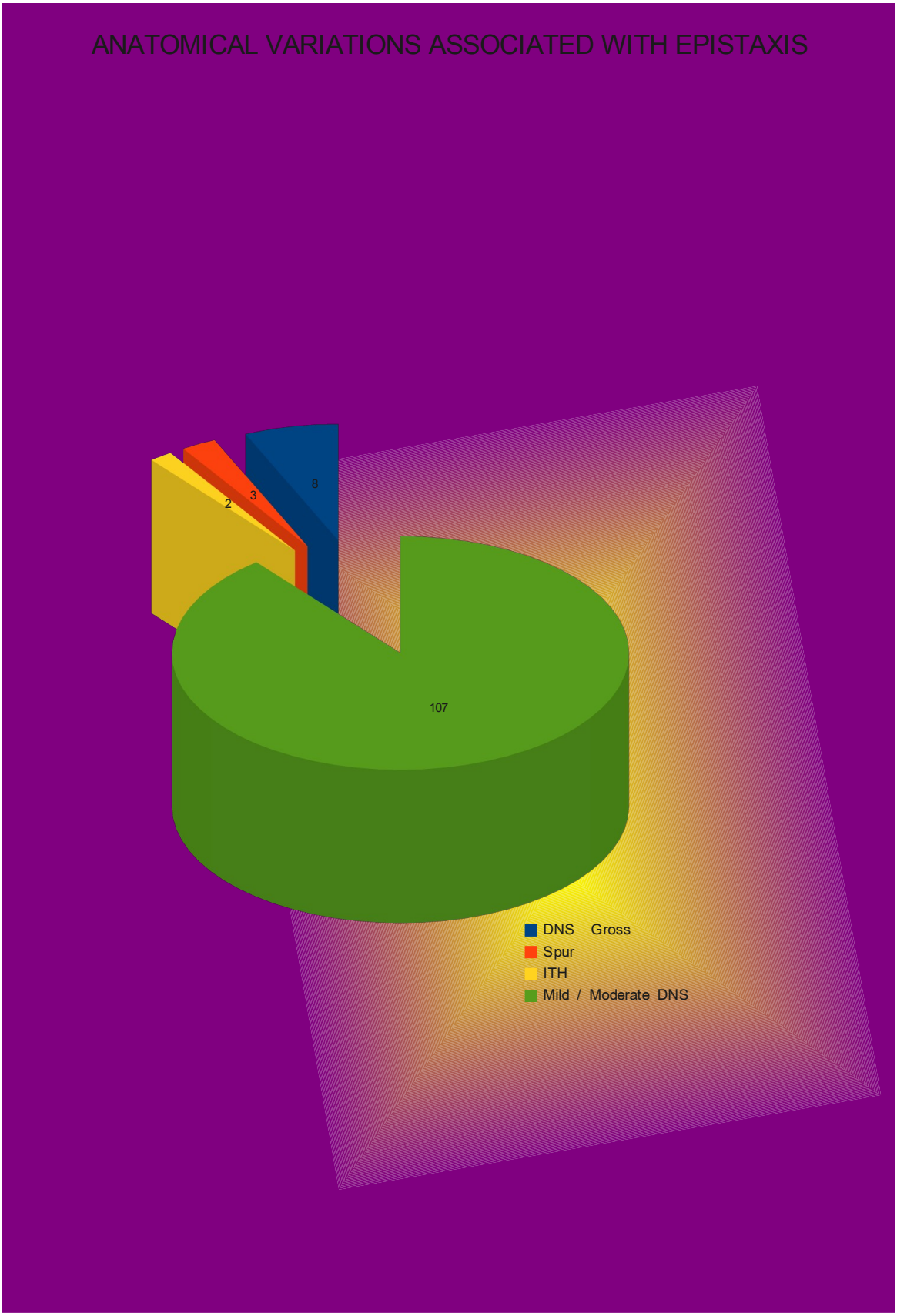
Table 7

Anatomical Variations	Nos.
DNS – Gross	8
Spur	3
ITH	2
Mild / Moderate DNS	107

Very few individuals have a perfectly straight septum. Mostly deviated nasal

septums are asymptomatic.

Septal abnormalities are common. Between 1 and 80% of the population have a significant deviation[37]. The perceived association between epistaxis and septal abnormalities could be coincidental.


ANATOMICAL VARIATIONS ASSOCIATED WITH EPISTAXIS
8
3
2
107
DNS Gross
Spur
ITH
Mild / Moderate DNS

ANTERIOR NASAL PACKING

ANTERIOR NASAL BLEEDING

ANTERIOR NASAL BLEEDING

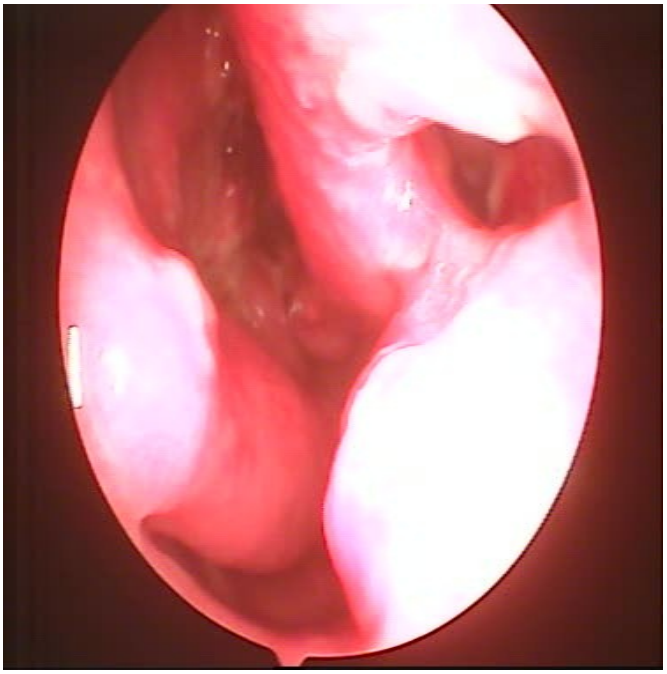
ANTERIOR NASAL PACKING

LITTLE'S AREA CONGESTION

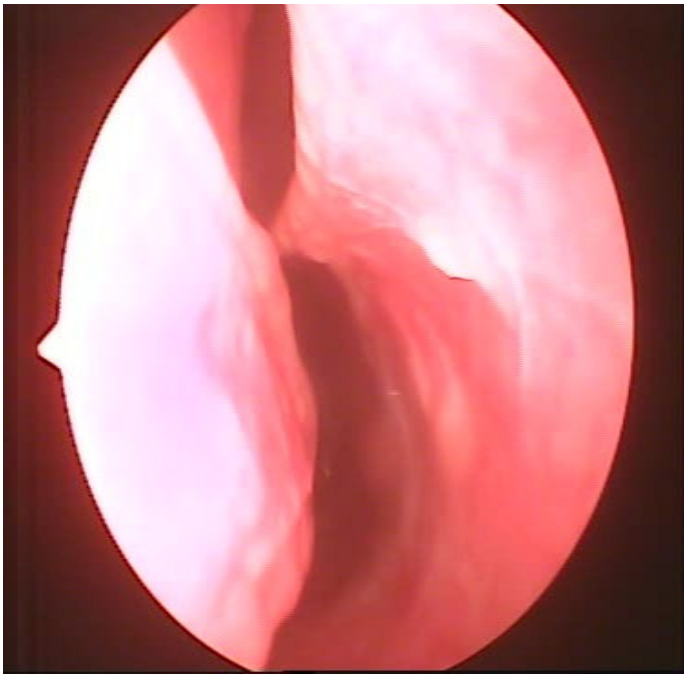
ANTERIOR NASAL CRUST

ANTERIOR NASAL CRUST

SEPTAL PERFORATION

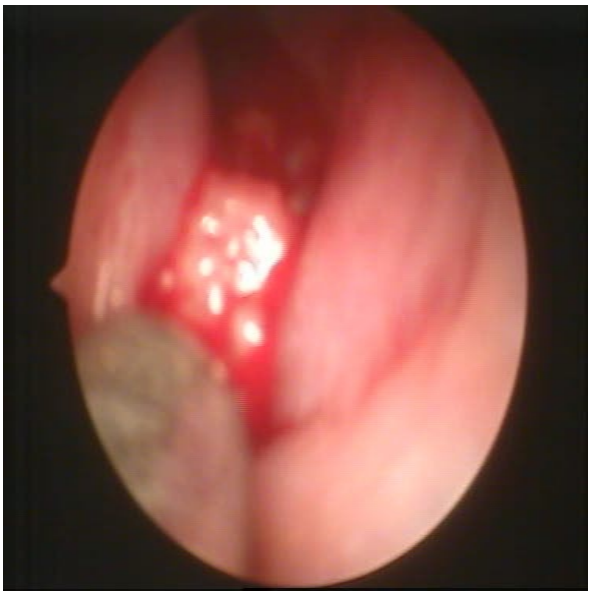


DNS WITH SPUR



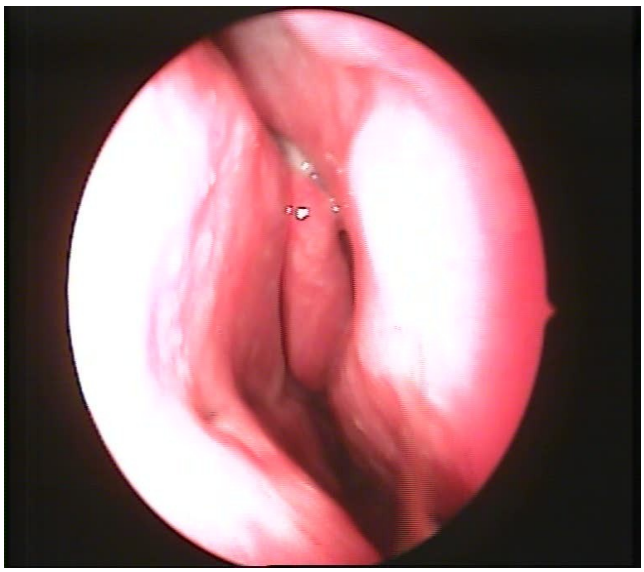
SEPTAL HAEMANGIOMA

RHINOSPORIDIOSIS

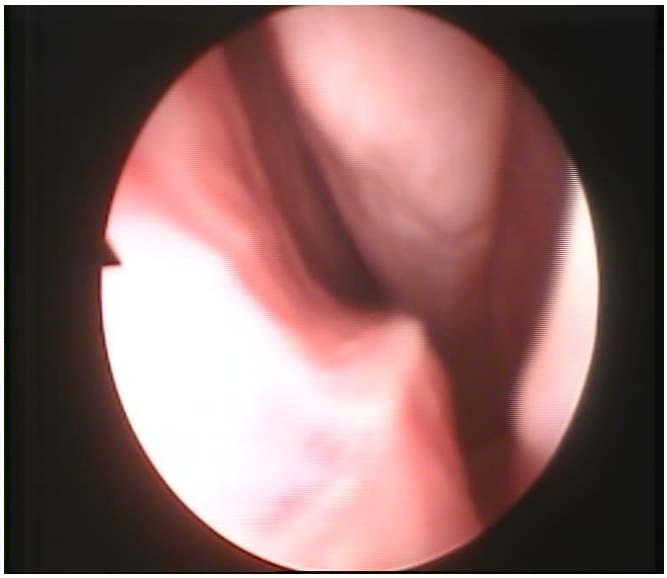


INVERTED PAPILLOMA

DNS WITH SINUSITIS



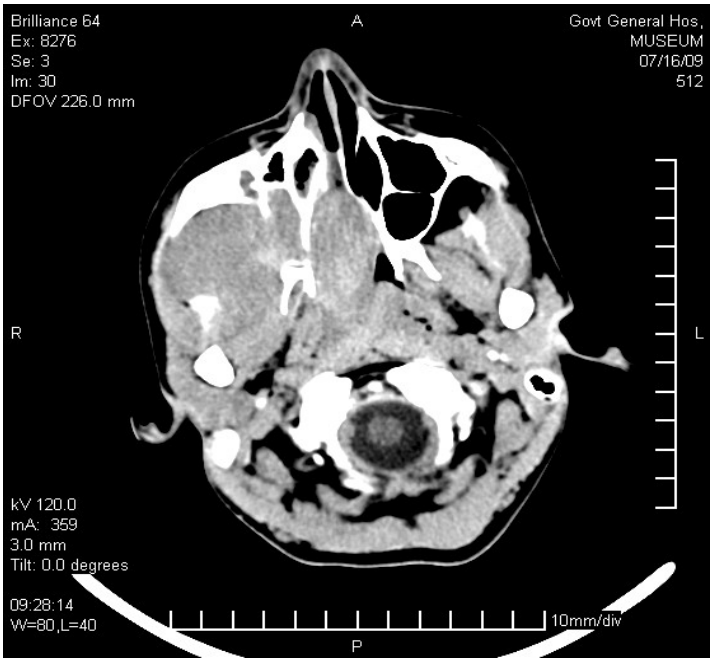
DNS WITH SPUR



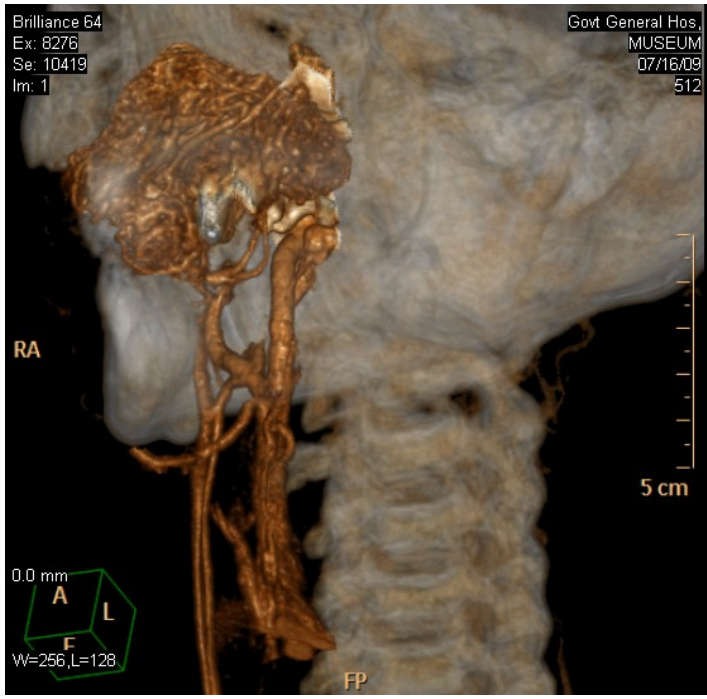
JNA PATIENT WITH ANTEIOR NASAL PACKING

AXIAL- CT SCAN

AXIAL- CT SCAN



CT ANGIOGRAM



MULTIPLE FINDINGS IN SINGLE PATIENT

Table 8

DNS with sinusitis	5
DNS with spur	3
JNA with DNS	5
Spur with Sinusitis	1
DNS with ITH	2

commonly noted finding is deviated nasal septum

Septal abnormalities are common. Between 1 and 80% of the population have a significant deviation. The perceived association between epistaxis and septal abnormalities could be coincidental.

BLEEDING DUE TO SYSTEMIC DISEASES

Table 9

Cause	Nos
Hypertension	6
Coagulation Deficits	3

Although hypertension has long been considered as a cause of epistaxis no causal relationship has been proved[14].

Hypertension can cause posterior bleeding and can cause severe bleeding in

older age group.

IATROGENIC CAUSES

Table 10

Cause	Nos
Post Septal correction	1
Post Reduction Fracture Nasal bone	1

Settled with conservative management with anterior nasal packing

SYMPTOMATOLOGY

Table 11

SYMPTOMS	NOs
Bleeding through nose	120
Nasal block	50
Head ache	39
Others	15

All the patients complained bleeding through nose of varying severity. Fifty patients had nasal block also with epistaxis. Thirty nine of the patients reported head ache with epistaxis.

Some patients had symptoms of allergy reporting sneezing and running nose.

One JNA patient had mild proptosis with swelling of cheek.

Two of the malignant growth maxilla patients also reported mild swilling of cheek.

NATURE OF BLEEDING

Table 12

Anterior Bleeding	75
Posterior Bleeding	33
Ant + Post Bleeding	12

Most of the patients had anterior nasal bleeding (75). Thirty three patients had posterior bleeding and twelve patients had both anterior and posterior bleeding.

In anterior epistaxis bleeding was from septum in 57 patients and bleeding was from lateral nasal wall in 18 patients.

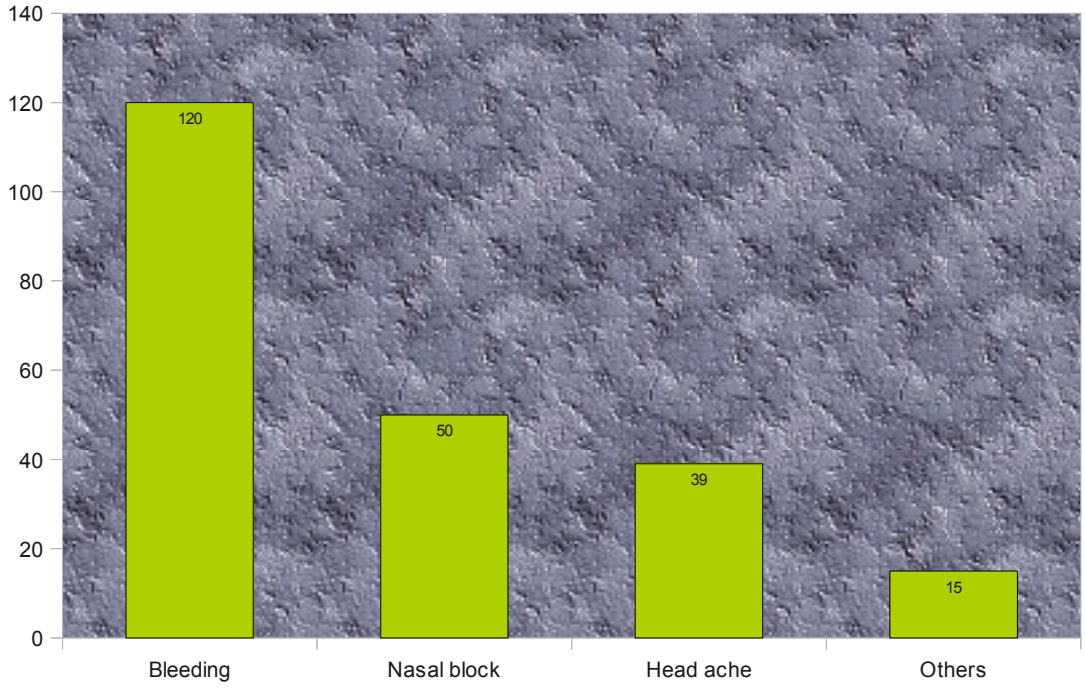
In posterior epistaxis 12 cases had bleeding from septum and 21 cases had bleeding form lateral nasal wall.

INVESTIGATION

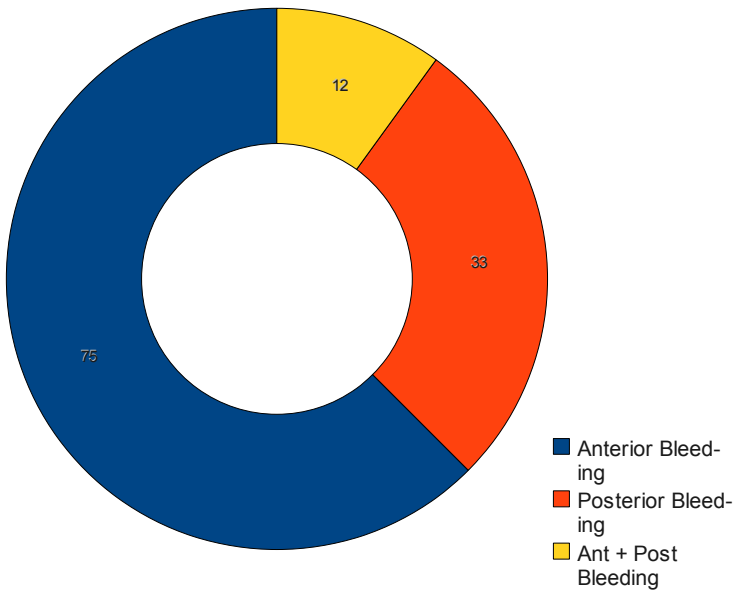
Table 13

Investigation	No's
Blood	120
DNE	120
X-Ray PNS	120
CT PNS	76
X-Ray NB	10

SYMPTOMATOLOGY



NATURE OF BLEEDING



Basic blood investigation was done to all the patients (Complete blood count, bleeding time, clotting time, blood urea, sugar, serum creatinine, serum electrolytes, blood grouping and typing). Complete bleeding profile investigation was done according to history, clinical suspicion and clinical diagnosis and based on other investigations.

Diagnostic nasal endoscopy was done to all the patients to identify the source of bleeding after removal of nasal packing if done prior.

X-ray PNS-water's view was taken to all the patients as a basic investigation. For those patients who had history of trauma X-ray nasal bone was also taken.

CT PNS was done to 76 patients and CT angiogram was done to one JNA patient.

ECG and CXR-PA view was done accordingly. MRI was taken when necessary.

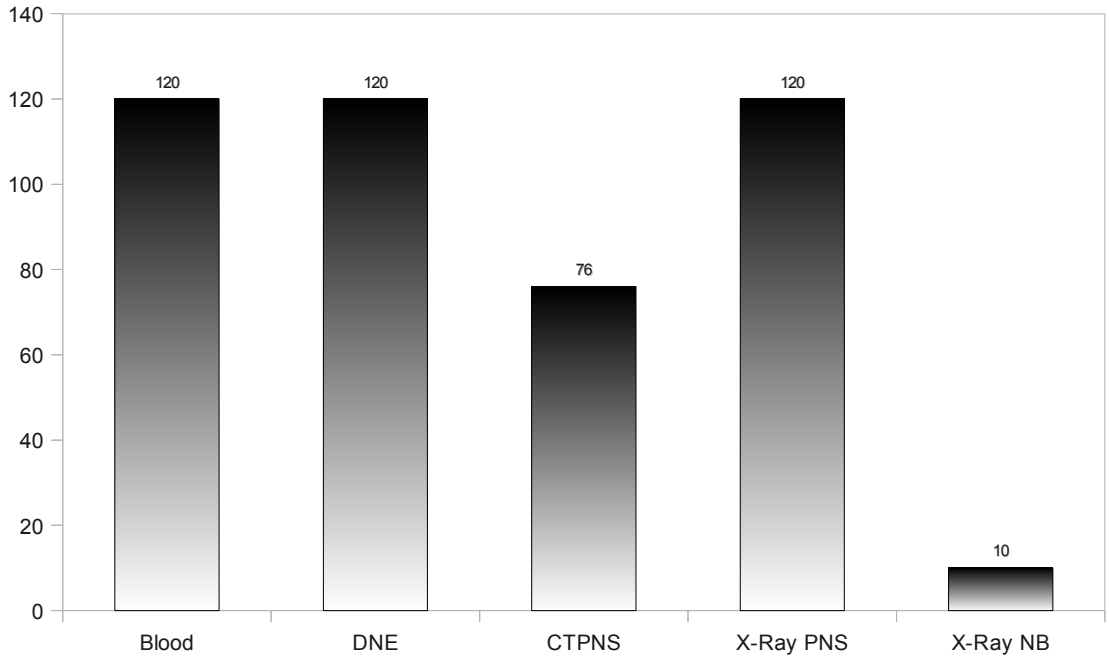
TREATMENT

Table 14

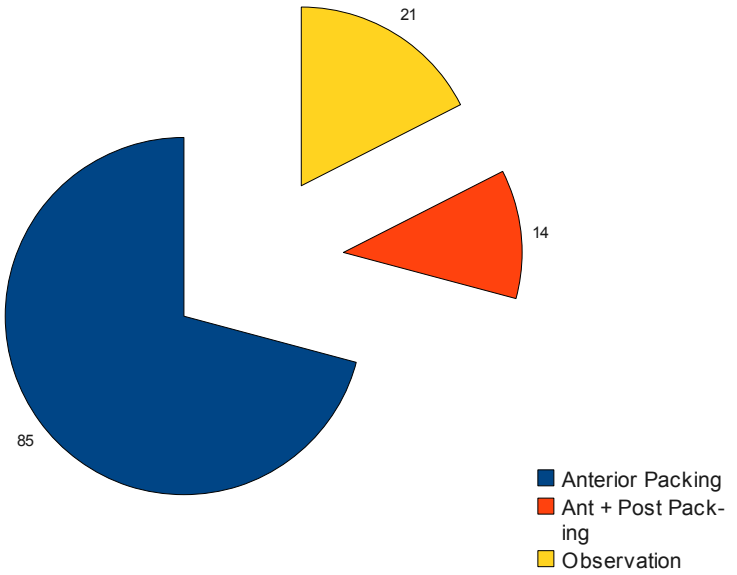
Nasal Packing	No's
Anterior Packing	85
Ant + Post Packing	14
Observation	21

Most of the patients were treated conservatively Anterior nasal packing was done to 85 patients and anterior and postnasal packing was done to 14 patients. 21 patients without active bleed during admission were kept under observation. Endoscopic sphenopalatine artery ligation was done to one patient and endoscopic cauterization of bleeding point and endoscopic cauterization of granulation tissue was done to two patients.

INVESTIGATIONS



TREATMENT



TREATMENT OF RELATED CAUSE

Table 15

Cause	Treatment	Nos
Chronic sinusitis	FESS	2
DNS with Chronic sinusitis	FESS with SMR	5
DNS	SMR	3
DNS with concha	SMR with conchaplasty	3
JNA	Endoscopic excision	8
JNA	Excision-Transantral	1
Rhinosporidiosis	Endoscopic excision	3
Septal Haemangioma	Endoscopic excision	1
Papilloma	Endoscopic excision	1
Sinonasal polyposis	Endoscopic polypectomy	6
Rhinolith	Endoscopic excision	1
Malignancy-Maxilla	Total maxillectomy	2
Malignancy-Maxilla	Medial Maxillectomy/Endoscopic debulking	1+1
Malignancy-Maxilla	Radio therapy	2
Fracture nasal bone	Reduction	7

DISCUSSION

Epistaxis- bleeding through the nose is one of the most common and most difficult emergencies to treat. About 60% of people experience the episode atleast once in life time with less than 10% of these requiring medical attention.

Most episodes are minor in nature but in some cases there could be massive bleeding. Epistaxis can be from anterior or posterior source and it can be from septum or lateral nasal wall. Both systemic and local factors play a role.

In the present study, 120 cases were studied from the out patient department and wards of the **upgraded institute of otorhinolaryngology of madras medical college and government general hospital Chennai.**

Aetiology :- In this study most cases were due to idiopathic cause (46.67%).

Our study correlates with

Stell[31]- Most cases are idiopathic, spontaneous bleeds without any proven precipitant or causal factor.

Pollice PA and Yoder MG[17].- Epistaxis.

Tan LKS and Calhoun KH[33]- Epistaxis.

Pond F and sizeland A[34]- Epistaxis.

Often no cause for the bleeding is identified.

Shaheen (1987)[21] has stated that majority of cases of epistaxis are idiopathic and this study also proves this.

Age group Distribution :- In the present study more commonly affected age group are 21 to 30, 13 to 20.

Next commonly affected age group is 51 to 60 and then 41 to 50

Shaheen showed in his study of age distribution of epistaxis an increase in frequency between ages 15 – 25 years and 45 – 65 years and this present study more or less correlates with that.

JNA occurs exclusively in adolescent males and this study also proves this

Sex Distribution :- In the present study males are commonly affected than females and this study more or less correlates with studies of

Juselius H. Epistaxis[13]

Juselies-: has stated that males are little more commonly affected and has also noted higher incidence of epistaxis in older age groups which more or less correlates with this study in which males are commonly affected than females and age group 41 to 60 years is the next commonly affected group

after 13 to 30 years age group

Padgham N Epistaxis[12] :- Study show male preponderance.

Pollice PA and Yoder MG[17] and **Rubin Grandis J, et al[32]:-** Epistaxis appears to occur more often in males than in females.

Tomkinson A and other[26] Males are slightly more affected than females.

In the present study most cases were anterior nasal bleeds and were managed conservatively with anterior nasal packing[24].

Juselius H. Epistaxis[13], Emanuel JM Epistaxis[2], O Donnell M Robertson G, McGarry GW[35]:- Most cases are easily manageable anterior nasal bleeds.

Surgical management were needed in 3 patients to control nasal bleeding.

Kotecha B, Fowler S, Harkness P, Walmsley J, Brown P, Topham J[36]:- Fewer than 10% of hospitalized patients require a general anaesthetic procedure to secure haemostasis.

Mild to moderate septal deviation were noted in most of the patients without any significant symptoms.

Roblin DG, Eccles R[37]:- Between 1 and 80% of the population have a significant deviation. Correlation between epistaxis and septal abnormalities can be coincidental

Investigations :- Basic blood investigation and diagnostic nasal endoscopy were done to all patients.

X-Ray nasal bone was taken for the patients with history of trauma to nose when nasal bone fracture was suspected clinically.

X-ray PNS was taken to all patients as a basic investigation to rule out any gross pathology.

CTPNS was taken to patients with clinical suspicion of malignancy, older age group patients, adolescent males with suspicion of JNA, patients of chronic sinusitis and to other required patients. MRI was taken when necessary.

ECG, chest X-ray, test for bleeding profile were taken to all relevant cases.

In older age groups radiological investigation is needed to rule out any malignancy.

Spontaneous, profuse bleeding in adolescent males requires nasal endoscopy, CT, MRI PNS to rule out JNA

Treatment :- Most cases were treated conservatively with anterior nasal packing(85 patients), some cases whom were without active bleeding during admission were observed(21 patients) and some cases were treated with anterior and posterior nasal packing(14 patients).

Endoscopic cauterization of suspected bleeding point was done to one patient and endoscopic cauterization of granulation tissue of septum was

done to one patient. Endoscopic sphenopalatine artery ligation was done to one patient.

Septal heamangioma and bleeding polyposis were treated endoscopically.

Other conditions like DNS, chronic sinusitis, rhinosporidiosis, nasal bone fracture, JNA, benign and malignant conditions were treated accordingly.

The endoscopy helps to identify bleeding points and to treat them effectively[4,6].

Recurrent bleeding was seen in eleven patients whom were controlled with conservative measures. Both previous episode and present episode were treated with anterior nasal packing.

SUMMARY

Epistaxis- bleeding through the nose is one of the most common and most difficult emergencies to treat. About 60% of people experience the episode atleast once in life time with less than 10% of these requiring medical attention.

Aims of this study is to find out the most common cause, early identification of the cause and thereby early management, prevalence in various age groups and sex and correlation of anatomical variations with respect to epistaxis.

In the present study, 120 cases were studied from the OPD and wards of the UIORL of MMC and GGH Chennai.

The commonest cause of epistaxis in both sexes was found to be idiopathic (46.67%). Other causes are trauma(8.33%), DNS with chronic sinusitis with spur (10.84%) and polyps (5%) malignancy (5%) . JNA is exclusively seen in adolescent males (5%). In older age group hypertension has to be considered and should be treated effectively.

Males (60%) were commonly affected than females (40%) in the present study.

Commonest age group involved was 21 to 30 years (27.5%), 13 to 20 years

(23.33%), 51 to 60 years (16.67%) and 41 to 50 years (15%).

Idiopathic as the commonest cause, males more commonly affected and age group involvement in the present study more or less correlates with other studies.

Most of the cases were anterior nasal bleed (62.5%) and were treated conservatively with anterior nasal packing.

Mild to moderate septal deviations was noted in most of the cases . These patients were not having any significant symptoms.

The incidence of recurrent epistaxis decreased significantly as most cases were diagnosed and treated immediately.

In older age groups radiological investigation was needed to rule out any malignancy.

Spontaneous, profuse bleeding in adolescent males requires nasal endoscopy, CT, MRI PNS to rule out JNA. Regular follow up was needed in post operative JNA cases rhinosporidiosis and in malignancy.

Moderate to severe epistaxis requires immediate packing and commonest problem in post packing nose is mucosal oedema and mucosal injury which may alter or mask the original pathology.

The endoscopy helps to identify bleeding points and to treat them effectively.

CONCLUSION

- Idiopathic is the most common cause of epistaxis in this present study ()46.67%.
- Other causes are trauma (8.33%), DNS with spurs and sinusitis (10.84%) and JNA in adolescent males.
- Males are commonly affected than females (60%).
- The most common age group affected is 13 to 30 years (50.83%) and 41 to 60 years (31.67%).
- Most cases are treated by conservative measures.

  In older age groups radiological investigation is needed to rule out any malignancy.
- Spontaneous, profuse bleeding in adolescent males requires nasal endoscopy, CT, MRI PNS to rule out JNA.

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PROFORMA

UPGRADED INSTITUTE OF OTORHINOLARYNGOLOGY
GOVERNMENT GENERAL HOSPITAL,
MADRAS MEDICAL COLLEGE,
CHENNAI – 3

Name Unit Age Sex

O.P. No, :
I.P. No :

Occupation

Address

Date of admission

Date of endoscopy

Date of surgery

Date of discharge

Presenting Complaints

Bleeding through the nose

Nasal block

Nasal discharg

Headache

Sneezing

Loss of smell

H/O hematemesis, melena, hemoptysis

H/O Ear, throat symptoms

H/O Loss of weight / Loss of appetite

Past History

H/o Previous episodes

H/o Foreign body

H/o Drug intake (oral, topical)

H/o Bleeding disorder

H/o Blood transfusion

H/o Systemic disorders – IHD, hypertension, diabetes, asthma

H/o Previous surgery.

Personal History

H/o Smoking,

H/o snuff use,

H/o Alcohol,

H/o Nose picking,

H/o Metallic poisoning.

Family History

H/o Bleeding diathesis

Treatment History

GENERAL EXAMINATION

Pulse rate,

Blood pressure,

Anemia,

Jaundice,

Cyanosis,

Clubbing,

Petechiae,

Ecchymosis,

Generalized lymphadenopathy,

Cardiovascular system,

Respiratory system,

Abdominal examination,

Examination of cranial nerves

ENT EXAMINATION

NOSE

External examination

External contour,
Bony tenderness,
Bony crepitus,
Columella retraction,
Other findings.

Anterior Rhinoscopy

Vestibule,
Little's area,
Mucosa,
Septum,
Turbinates,
Bleeding points / mass,
Other findings

Posterior Rhinoscopy

Choana,
Posterior end of turbinates,
Roof of nasopharynx,
Eustachian tube orifice,
Bleeding points,
Adenoids / mass,
Other findings.

Ear :

Throat :

Neck :

DIAGNOSTIC NASAL ENDOSCOPY

		Right	Left
Little's area			
Turbinoseptal classification			
Inferior matus			
	Woodruff's area		
	Other findings		
Inferior turbinate			
Septum	DNS		
	Spur		
Middle turbinate			
Middle meatus			
Uncinate			
Bulla			
Aggernasi			
Other findings			

	Right	Left
High septal deviation		
Superior turbinate		
Spheno ethmoid recess		
Apex of nasal fossa		
Choana		
Nasopharynx		
Eustachian tube orifice		
Roof of nasopharynx		
Fossa of Rosenmuller		
Other findings		

OTHER INVESTIGATION

Complete haemogram,

Packed cell volume,

Blood grouping & typing,

Bleeding profile,

Liver function tests,

Blood urea / sugar,

Chest X-ray,

ECG,

X-ray paranasal sinuses – Water's view,

CT PNS – coronal and axial cuts → 5mm → plain and contrast films,

Angiogram / Brain,

Digital substraction angiogram,

MRI – PNS / Brain / Angiogram

EXPERT OPINION

Haematologist opinion

Physician opinion

DIAGNOSIS:

ABBREVIATIONS

DNS – Deviated Nasal Septum.

JNA – Juvenile Nasopharyngeal Angiofibroma

Chr.Sinusitis – Chronic Sinusitis

ITH – Inferior Turbinate Hypertrophy

DNE – Diagnostic Nasal Endoscopy

ESPAL – Endoscopic Sphenopalatine Artery Ligation

PCV – Packed Cell Volume

EACA – Epsilon Amino Caproic Acid

BIPP – Bismuth Iodoform Paraffin Paste

Inv. Papilloma – Inverted Papilloma

FESS – Functional Endoscopic Sinus Surgery

SMR – Sub Mucosal Resection

Endo. Excision – Endoscopic Excision

ECA – External Carotid Artery

NB – Fracture Nasal Bone

IHD – Ischaemic Heart Disease

MASTER CHART

Sl. No	Name	Age	Sex	I.P No. / O.P. No.	Symptomatology				Investigations				Diagnosis	Treatment	
					Blee Ding	Nasal Block	Head Ache	Oth Ers	Blood	DNE	X-Ray PNS	C.T PNS		Primary	Related
1	John Lokesh	26	M	70983	+				√	√	√		Idiopathic	AN packing	
2	Ramesh	25	M	63630	+	+	+	+	√	√	√	√	DNS / Chr. Sinusitis	Observation	FESS / SMR
3	Isski	27	M	27961	+	+	+	+	√	√	√	√	DNS / Chr. Sinusitis	AN Packing	FESS / SMR
4	Ethiraj	22	M	44871	+				√	√	√		Iatrogenic (# N.B – Reduction)	AN packing	
5	Sanjay Kumar	28	M	64934	+				√	√	√	√	Idiopathic	AN packing	
6	Ramalingam	24	M	57548	+	+			√	√	√	√	JNA	AN + PN Packing	Endo. Excision
7	Veera	24	M	53684	+	+			√	√	√	√	Bleeding Polyposis Rt Nasal Cavity	AN Packing	Excision
8	Thangamani	30	M	55142	+				√	√	√		Idiopathic	Observation	
9	Gunasakaran	33	M	65122	+	+			√	√	√		Idiopathic	AN packing	
10	Nadarajan	55	M	14116	+		+		√	√	√	√	Idiopathic	AN packing	
11	Mani	57	M	60297	+		+		√	√	√	√	Hypertensive	AN + PN Packing	
12	Jayapragadeesh	19	M	71032	+	+	+		√	√	√		Iatrogenic (Post Septal Correction)	AN packing	
13	Charles	18	M	74261	+				√	√	√		Trauma # N.B	Observation	Reduction
14	Saravanan	32	M	82751	+	+	+		√	√	√		Trauma # N.B	AN packing	Reduction
15	Salman	35	M	3128	+	+			√	√	√		Trauma # N.B	Observation	Reduction
16	Vadivel	20	M	18197	+				√	√	√		RTA	AN packing	
17	Raj	38	M	56060	+				√	√	√		RTA	Observation	
18	Munusami	70	M	45065	+		+		√	√	√	√	Idiopathic	AN packing	
19	Sunder	45	M	64840	+				√	√	√		Septal Perforation	Observation	
20	Subramani	56	M	65588	+	+	+	+	√	√	√	√	CA. Maxilla – LT	AN packing	LT. Total Maxillectomy
21	Shakulhamidh	62	M	84464	+	+	+		√	√	√	√	CA. Maxilla – LT	Observation	Endo. Debulking
22	Vijayan	52	M	86105	+	+	+	+	√	√	√	√	CA. Maxilla – LT	Observation	LT. Total Maxillectomy
23	Rajendran	54	M	44487	+				√	√	√	√	Idiopathic	AN + PN Packing	
24	Perumal	50	M	53870	+	+			√	√	√	√	Idiopathic	AN packing	
25	Munusami	55	M	64333	+	+	+	+	√	√	√	√	Idiopathic	AN + PN Packing	
26	Muthu	47	M	49989	+				√	√	√	√	Idiopathic	AN packing	
27	AllahaPichai	50	M	35317	+				√	√	√	√	Hypertensive	AN packing	
28	Subramani	46	M	30762	+				√	√	√	√	Hypertensive	AN packing	
29	Pavalamalai	50	M	49982	+	+	+	+	√	√	√	√	Hypertensive	AN packing	
30	Muthu	47	M	49989	+				√	√	√	√	Idiopathic	AN packing	

SI. No	Name	Age	Sex	I.P No. / O.P. No.	Symptomatology				Investigations				Diagnosis	Treatment	
					Blee Ding	Nasal Block	Head Ache	Oth Ers	Blood	DNE	X-Ray PNS	C.T PNS		Primary	Related
31	Purushothaman	25	M	71013	+	+			√	√	√		Idiopathic	AN packing	
32	Shameer	28	M	74042	+				√	√	√		Idiopathic	AN packing	
33	Subramani	65	M	76952	+				√	√	√	√	Idiopathic	AN packing	
34	Gogulraj	14	M	66092	+				√	√	√	√	Idiopathic	AN packing	
35	Velu	55	M	70329	+				√	√	√	√	Idiopathic	AN packing	
36	Johnpal	28	M	87424	+	+	+	+	√	√	√		Idiopathic	AN packing	
37	Kannan	38	M	82712	+				√	√	√		Idiopathic	Observation	
38	Sudhaher	35	M	90767	+				√	√	√		Idiopathic	AN packing	
39	Boopalan	42	M	90777	+				√	√	√	√	Idiopathic	AN packing	
40	Palani	25	M	96511	+				√	√	√		Idiopathic	AN packing	
41	Deva	46	M	61592	+		+	+	√	√	√	√	Idiopathic	AN packing	
42	Mohan	48	M	7586	+				√	√	√	√	Polycythemia	AN packing	
43	Jeevan	30	M	75685	+				√	√	√		Idiopathic	AN packing	
44	Subramani	17	M	1904	+				√	√	√	√	Idiopathic	AN packing	
45	Ganesh	48	M	84575	+				√	√	√	√	Haematological	AN + PN Packing	
46	Masthan	30	M	5178	+				√	√	√		Idiopathic	Observation	
47	Radhakrishnan	58	M	7230	+				√	√	√	√	Idiopathic	AN packing	
48	Vengadesan	38	M	5403	+				√	√	√		Idiopathic	AN packing	
49	Richard	32	M	9213	+	+			√	√	√		Idiopathic	AN packing	
50	Munusami	62	M	4057	+				√	√	√	√	Idiopathic	AN packing	
51	Raja	48	M	15493	+				√	√	√	√	Clotting factor Dysfunction	DNS / Chr. Sinusitis	DNS / Chr. Sinusitis
52	Senkuttuvan	37	M	10303	+				√	√	√		Idiopathic	Observation	
53	Jegan	19	M	22364	+				√	√	√	√	Idiopathic	AN packing	
54	Mohamed Shameer	22	M	14470	+				√	√	√		Trauma	AN packing	
55	Ashraf	25	M	1951	+	+			√	√	√	√	DNS / Spur	AN packing	SMR
56	Subramani	30	M	86006	+	+	+		√	√	√	√	DNS Rt ITH	Observation	SMR /Inf. Turbinectomy
57	Sivasubramaniam	53	M	60014	+	+			√	√	√	√	Rhinosporidiosis / Rt	AN packing	Endo. Excision
58	Rajagopal	16	M	72656	+	+			√	√	√	√	REC. Rhinosporidiosis Lt	AN packing	Endo. Excision
59	Kumar	32	M	74302	+	+	+		√	√	√	√	REC. Rhinosporidiosis Lt	Observation	Endo. Excision
60	Raghman	55	M	52232	+	+			√	√	√	√	Adenoca Rt Maxilla	Observation	Rt Medial Maxillectomy

Sl. No	Name	Age	Sex	I.P No. / O.P. No.	Symptomatology				Investigations				Diagnosis	Treatment	
					Blee Ding	Nasal Block	Head Ache	Oth Ers	Blood	DNE	X-Ray PNS	C.T PNS		Primary	Related
61	Tirupathi	55	M	46263	+		+	+	√	√	√	√	REC. Lt Maxillary Growth	AN + PN Packing	
62	Balasundaram	18	M	53301	+	+	+		√	√	√	√	REC. JNA	AN + PN Packing	Endo. Excision
63	Kaliyan	20	M	65561	+	+			√	√	√	√	REC. JNA	AN packing	Endo. Excision
64	Rajkumar	20	M	79342	+	+	+	+	√	√	√	√	REC. JNA	AN + PN Packing	Endo. Excision
65	Deelipan	18	M	84409	+	+	+		√	√	√	√	JNA	AN + PN Packing	Endo. Excision
66	Amarnath	16	M	4381	+	+			√	√	√	√	JNA	AN + PN Packing	Endo. Excision
67	Nadarajan	20	M	14945	+	+	+		√	√	√	√	REC. JNA	AN + PN Packing	Endo. Excision
68	Vijay	19	M	29183	+	+			√	√	√	√	JNA	AN + PN Packing	Endo. Excision
69	Ilango	17	M	51188	+	+	+	+	√	√	√	√	JNA	AN packing	Transantral / ECA Contorl
70	Murugan	50	M	68081	+				√	√	√		Granulation Tissue Lt Septum	AN packing	Cauterization
71	Panneer selvam	16	M	50310	+				√	√	√		Septal Haemangioma - Rt	AN packing	Excision
72	Murugan	14	M	16633	+	+	+		√	√	√	√	Rhinolith	AN packing	Endo. Excision
73	Ashadevi	15	F	48745	+				√	√	√		Idiopathic	AN packing	
74	Meenachi	51	F	75636	+				√	√	√	√	Idiopathic	AN packing	
75	Kamachi	20	F	16413	+				√	√	√		Idiopathic	AN packing	
76	Pattu roja	34	F	23496	+			+	√	√	√	√	Idiopathic	AN packing	
77	Vijaya lakshmi	31	F	91779	+				√	√	√		Idiopathic	Observation	
78	Basheera	20	F	14634	+	+			√	√	√		Idiopathic	AN packing	
79	Alzhammal	55	F	31772	+		+		√	√	√	√	Hypertensive	AN packing	
80	Geetha	16	F	39655	+				√	√	√		Idiopathic	AN packing	
81	Latha	42	F	45151	+				√	√	√	√	Idiopathic	AN packing	
82	Nagarathinam	52	F	57234	+				√	√	√	√	Idiopathic	AN packing	
83	Vanaroja	20	F	47103	+				√	√	√		Idiopathic	AN packing + Cauterization	
84	Bakkiyam	60	F	39937	+				√	√	√	√	Hypertensive	AN packing	
85	Sudha	35	F	25301	+	+			√	√	√		Trauma # N.B	Observation	Reduction
86	Nathiya	19	F	18719	+				√	√	√		Idiopathic	AN packing	
87	Ravathi	25	F	13351	+				√	√	√		Idiopathic	AN packing	
88	Ulakammal	60	F	63714	+		+		√	√	√	√	Idiopathic	AN packing	
89	Vino	20	F	67225	+				√	√	√	√	Idiopathic	AN packing	
90	Mohamad beham	55	F	70112	+	+			√	√	√	√	Papilloma Lt	AN packing	Excision

SI. No	Name	Age	Sex	I.P No. / O.P. No.	Symptomatology				Investigations				Diagnosis	Treatment	
					Blee Ding	Nasal Block	Head Ache	Oth Ers	Blood	DNE	X-Ray PNS	C.T PNS		Primary	Related
91	Eswari	25	F	68191	+				√	√	√		Trauma # N.B	Observation	Reduction
92	Priya	21	F	77301	+				√	√	√		Trauma # N.B	AN packing	Reduction
93	Barathi	25	F	67667	+	+	+	+	√	√	√	√	DNS / Concha Bullosa	AN packing	SMR / Conchaplasty
94	Lalitha	34	F	40971	+	+	+		√	√	√	√	DNS / Bil.ITH	Observation	SMR / Turbinectomy
95	Nagalakshmi	33	F	45154	+	+			√	√	√		DNS / Spur	AN packing	SMR
96	Vasantha	60	F	72230	+				√	√	√	√	Idiopathic	AN packing	
97	Samakhani	49	F	80121	+	+			√	√	√	√	REC. Inverted Papilloma	AN packing	
98	Kanchana	24	F	61029	+	+	+		√	√	√	√	DNS / Bil.Concha / Chr. Sinusitis	AN packing	FESS / Conchaplasty / SMR
99	Priya	18	F	57968	+	+			√	√	√		DNS / Spur	AN packing	SMR
100	Radha	30	F	50066	+	+	+		√	√	√	√	DNS / Chr. Sinusitis	AN packing	FESS / SMR
101	Kavitha	20	F	40903	+	+	+	+	√	√	√	√	Chr. Sinusitis	Observation	FESS
102	Suganthi	24	F	18253	+	+	+	+	√	√	√	√	Chr. Sinusitis	AN packing	FESS
103	Pushpavalli	38	F	55830	+	+	+		√	√	√	√	DNS / Chr. Sinusitis	AN packing	FESS / Septoplasty / Turbinoplasty
104	Bhuvaneswari	28	F	73556	+	+	+		√	√	√	√	Sinonasal Polyposis	AN packing	Endo. Polypectomy
105	Seethammal	54	F	74458	+	+	+		√	√	√	√	AntraChoanal Polyp Lt	Observation	Endo. Polypectomy
106	Vennila	45	F	72706	+	+	+		√	√	√	√	Bil.Ethmoidal Polyposis	AN packing	Endo. Polypectomy
107	Priya	18	F	50113	+				√	√	√		Idiopathic	AN packing	
108	Nirmala	26	F	53612	+				√	√	√		Idiopathic	AN packing	
109	Anjalai	25	F	55318	+				√	√	√		Idiopathic	AN packing	
110	Mari	48	F	46068	+	+	+		√	√	√	√	Bleeding Polyposis	AN packing	Endo. Polypectomy
111	Chithra	24	F	32733	+				√	√	√	√	Idiopathic	AN packing	
112	Lakshmi	25	F	32746	+				√	√	√	√	Idiopathic	AN packing	
113	Parimala	31	F	75226	+				√	√	√	√	Idiopathic	AN + PN packing + Endo. Cauterization - SPA	
114	Muniyammal	60	F	57044	+				√	√	√	√	Idiopathic	AN packing	
115	Meera	20	F	84278	+				√	√	√		Idiopathic	AN packing	
116	Gandimathi	50	F	13356	+				√	√	√	√	Idiopathic	AN packing	
117	Umadevi	22	F	1548	+				√	√	√		Idiopathic	AN packing	
118	Janath	27	F	56286	+				√	√	√		Trauma # N.B	AN packing	Reduction
119	Rajakumari	45	F	53045	+		+		√	√	√	√	CA. Maxilla	Observation	Rediotheraphy
120	Mallika	50	F	58116	+	+	+		√	√	√	√	Lt. Nasal mass	AN packing	Endo. Excision